HISTOPATHOLOGICAL SPECTRUM OF NON NEOPLASTIC LIVER DISEASES

A THESIS SUBMITTED TO THE BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT (PUNJAB)

FOR THE AWARD OF DEGREE OF

DOCTOR OF MEDICINE

IN

PATHOLOGY

BY

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ACKNOWLEDGEMENT

Words cannot express the feeling of deepest regard and sense of gratitude towards all those people who have helped me carry out the ritual of writing thesis, because without their guidance, this thesis wouldn't have seen the light.

With great regard and with due respect, I acknowledge the boundless affection and the help I received from my respected guide ***Dr. Neena Sood*** *(Former Professor & Head, Department of Pathology, DMCH, Ludhiana) for her masterly advice, sustained encouragement, generous help and invaluable guidance throughout the course of the study. Her immense knowledge and incredible ability to teach was a tremendous help to me. I shall always regard her as an inexhaustible source of inspiration for the rest of my life.*

I am highly indebted to ***Dr. Aminder Singh*** *(Associate Professor, Department of Pathology, DMCH, Ludhiana), my co-supervisor who was instrumental in guiding the study. His constant supervision and words of inspiration have contributed largely to the successful completion of this study.*

Words fail to express my profound pleasure and deep sense of gratitude to my esteemed teacher and co-supervisor, ***Dr. Ajit Sood*** *(Professor and Head, Department of Gastroenterology, DMCH, Ludhiana) for his constant support, academic guidance and constructive criticism, which helped me delve deeper into the study. His unending enthusiasm was invaluable at every stage of the study.*

I am also indebted to ***Dr Sandeep Puri,*** *Principal, DMCH, Ludhiana for providing all the necessary facilities in the department.*

With great regards and due decorum, I acknowledge my sincere thanks to ***Dr. Harpreet Kaur****, Professor and Head* ***Dr. Bhavna Garg****, Professor and* ***Dr. Pavneet Kaur Selhi,*** *Professor, Department of Pathology, DMCH, Ludhiana.*

I owe a debt of gratitude to my respected teachers ***Dr. Vikram Narang, Dr. Sumit Grover, Dr. Ruchita Tayagi and Dr. Ankita Soni*** *for guiding and encouraging me throughout the period of study.*

"Behind every daughter who believes in herself, are the parents who believed first." With this saying, I am eternally indebted to my parents ***Dr. Rajinder Gupta*** *and* ***Mrs. Manju Gupta*** *for their valuable support that I have received throughout my life and for believing in me. I place all the credit of my success and achievements in their holy feet. They are the ultimate role models for me. I thank god for giving me the best parents in the world.*

I am grateful to my brother ***Dr. Raghav Gupta*** *for being my pillar of strength at all times.*

I would like to thank ***Dr. Yuganshu Gupta*** *for his immense insight, expertise, and scientific inputs. Without his unconditional support, constant motivation, and cooperation at every step of this journey, this thesis would have not been possible.*

I am indebted to my seniors ***Dr. Saveena Jindal, Dr. Heena, Dr. Akriti Jindal, Dr. Sonia, Dr. Aman kaur, Dr. Aditi Sharma*** *and fellow colleagues* ***Dr. Aastha Sharma, Dr. Anshul Jain, Dr. Harsimran Kaur and Dr. Shefali Mehmi*** *for their ever readiness to help me during the study. I also take this opportunity to express my gratitude to my juniors* ***Dr. Gagandeep Singh, Dr. Rashi Aggarwal, Dr. Jasleen Kaur, Dr. Sargun, Dr. Kajal, Dr. Parul, Dr. Devika, Dr. Mehar, Dr. Ramneek and Dr. Nishav.***

I would also like to thank all the technical staff of my department for their constant help and encouragement.

I would like to mention the constant support and encouragement of my friends ***Dr. Kshish Aggarwal, Dr. Prateet Kaur, Dr. Gourav Janweja*** *and* ***Dr. Nikhar Somani*** *without the support of you all it would have really been a difficult task.*

Last but not the least I would like to thank ***Mrs. Namita Bansal,***

Miss. Neha Bhatt, Mr. Naveen Bhatia *and all the R&D staff for their skillful analysis & typing of the manuscript.*

I acknowledge the help of all those invisible hands who are responsible for the successful outcome of this dissertation.

Last but not the least I would also like to pay my immense gratitude and respect to God almighty for his blessings, affection and energy provided to me to complete my thesis work well in time.

Dr. Navita Gupta

ABBREVATIONS

AB	Absent
A/G	Albumin/Globulin
ALF	Acute Liver Failure
ATP	Adenosine Triphosphate
ATT	Antitubercular Therapy
ALP	Alkaline Phosphatase
ANA	Antinuclear Antibodies
ASMA	Anti Smooth Muscle Antigen
Anti SLA/ LP	Anti Soluble Liver Antigen/ Liver Pancreas Antigen
AMA	Anti Mitochondrial Antibodies
ANCA	Antinuclear Cytoplasmic Antibody
Anti- LKM 1	Anti Liver Kidney Microsome 1
AST	Aspartate Transaminase
ALT	Alanine Transaminase
ABCC2	ATP Binding Cassette sub-family C member 2
ALD	Alcoholic Liver Disease
AH	Alcoholic Hepatitis
AIH	Autoimmune Hepatitis
BDL	Bile Duct Lesion
BRIC	Benign Recurrent Intermittent Cholangitis
CC	Cryptogenic Cirrhosis
CRN	Clinical Research Network
CLD	Chronic Liver Disease
CVH	Chronic Viral Hepatitis
DILI	Drug Induced Liver Injury
DNA	Deoxy ribonucleic Acid
DJS	Dubin Johnson Syndrome
DALY	Disability Adjusted Life Years
GGT	Gamma Glutamyl Transpeptidase
HCV	Hepatitis C Virus
HBV	Hepatitis B Virus
HAV	Hepatitis A Virus
HEV	hepatitis E Virus
HCC	Hepatocellular Carcinoma
HSC	Hepatic Stellate Cells
LFT	Liver Function Test
LF	Lymphoid Follicles
LA	Lymphoid Aggregates
MRP2	Multidrug Resistance- associated Protein 2
ND	Not Done
NR	Non-Reactive

NP	Not Present
NSAID	Non-Steroidal Anti-Inflammatory Drugs
NAFLD	Nonalcoholic Fatty Liver Disease
NASH	Nonalcoholic Steatohepatitis
PBC	Primary Biliary Cirrhosis
PSC	Primary Sclerosing Cholangitis
PFIC	Progressive Familial Intrahepatic Cholestasis
PAS	Periodic Acid Schiff
PHA	Primary Hepatic Amyloidosis
PCR	Polymerase Chain Reaction
P	Present
RNA	Ribonucleic Acid
UGT	Glucuronyltransferase
ULN	Upper Limit of Normal
UR	Unremarkable
ZN	Zeil Nelson

ABSTRACT

Introduction: There is a wide spectrum of non-neoplastic liver diseases. Histopathological diagnosis of liver biopsies is done to establish the diagnosis, for staging and/or prognostication, and for treatment planning. Liver histology, clinical and laboratory data are used in conjugation to make the diagnosis. Liver diseases and cirrhosis contribute to significant mortality and morbidity involving worldwide and India.

Aim: To study histopathological spectrum of non-neoplastic liver diseases.

Methods: The present 5 years {1.5 year prospective analysis (January 2019 – June 2020) and 3.5 year retrospective analysis (July 2015 – December 2018)} study of trucut liver biopsies was conducted in the Department of Pathology, Dayanand Medical College and Hospital, Ludhiana. During this period a total of 364 cases were analysed. The clinical, biochemical and radiological findings were recorded according to the proforma.

Results: During the study period a total of 364 cases having liver pathology which were then analysed and classified on the basis of aetiology. There were 11 cases in which no specific pathology was found. Out of the remaining 353 cases, majority cases had metabolic cause of liver injury which constituted 29.2% of the total. Infectious causes were seen in 23.2% cases which included viral hepatitis and tuberculosis. It was seen that 19.2% cases had immune mediated causes and autoimmune hepatitis is the leading aetiology in this category. 9.9% cases had drug and toxin induced hepatotoxicity. Infiltrative disorders were seen in 5.1% cases. Mechanical causes were seen in 4.2% cases. Miscellaneous causes were seen in 8.3% cases.

Conclusion: Histopathologic examination is essential to analyse the etiopathogenesis of liver injury in trucut liver biopsies.

Keywords: Trucut liver biopsy, chronic liver diseases, Cirrhosis, Non-alcoholic liver diseases.

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INTRODUCTION

Percutaneous, ultrasound-guided liver biopsy remains the most reliable way to diagnose liver diseases because the method is simple, rapid, inexpensive, and quite safe[1]. Sometimes wedge liver biopsies are taken for unexpected lesions encountered during surgery. For the proper grading and staging of chronic liver diseases the needle specimen should be at least 1 cm long comprising a minimum of 8–12 portal tracts[2]. As the standard liver biopsy is only 1/50,000 of the liver tissue sampling error is source of possible diagnostic problems. Many diseases, such as primary biliary cirrhosis, autoimmune hepatitis or primary sclerosing cholangitis involve the liver in an unpredictable manner and are often presenting as patchy or sporadic involvement, which might not be included in the random biopsy. Clinico-pathologic correlation is the only way to avoid misinterpretation of pathologic findings. If the pathologic findings do not correlate with the clinical data, a comment deserves to be made about such a discrepancy. Additional samples or a clinical follow up is helpful in most of the cases.

Liver biopsies are done to establish the diagnosis, for staging and/or prognostication, and for treatment planning. The various diseases like viral hepatitis and non-viral liver diseases like autoimmune hepatitis, autoimmune cholestasis, drug induced liver injury, fatty liver diseases related to alcoholic and nonalcoholic causes, metabolic diseases like hemochromatosis, Wilson's disease, alpha 1 antitrypsin deficiency require histopathological correlation. Biopsies are also sometimes performed for acute liver failure, tuberculosis, sarcoidosis, liver transplantation, rejection and reinfection, solitary and multiple non neoplastic focal hepatic lesions.

For patients with viral hepatitis, liver biopsy is used for grading, staging and for treatment monitoring over the course of disease. Hepatitis A and E are the causes of acute hepatitis and hepatitis B, C, D have the potential to cause chronic disease. The inflammatory cells in both acute and chronic viral hepatitis are mainly T lymphocytes. Hence it is the pattern of injury that is different between the two-time courses, not

the nature of infiltrate. Hepatitis C virus (HCV) is a globally prevalent pathogen and is a major cause of healthcare burden in India. HCV poses a significant problem in the state of Punjab, India due to the higher prevalence of risk factors like unsafe medical practices (including unsafe injections and dental procedures) and intravenous drug use. In patients with cholestatic liver diseases, liver biopsy is a valuable means of detecting or ruling out the autoimmune disorders when the laboratory findings are not conclusive. Histological examination helps to differentiate alcoholic from non-alcoholic fatty-liver disease (NAFLD) and in assessment of inflammation and degree of fibrosis. There are three forms of alcoholic liver injury namely hepatocellular steatosis, alcoholic steatohepatitis and steatofibrosis. NAFLD is a spectrum of disease ranging from simple steatosis, to non-alcoholic steatohepatitis (NASH), through to advanced fibrosis and cirrhosis. The majority have simple steatosis, but approximately 10–30% develop NASH. It is associated with a poor long-term prognosis. When the laboratory findings are inconclusive, liver biopsy can provide the initial diagnosis of storage diseases and metabolic diseases of liver. Drug-induced liver injury (DILI) is the most common cause of acute liver failure (ALF) in the western world. Except paracetamol overdose, almost all DILI encountered in the clinical setting is idiosyncratic in nature because affected individuals represent only a small proportion of those treated with such drugs. Antibiotics are the class of drugs most frequently associated with idiosyncratic DILI, although recent studies indicate that herbal and dietary supplements are an increasingly recognised cause. Among all diagnostic methods for hepatic nodules, liver biopsy has the greatest sensitivity and specificity with respect to the determination of malignancy. Liver-cell adenoma is sometimes difficult to distinguish from well-differentiated hepatocellular carcinoma. In such cases, a biopsy is advisable if the imaging studies are inconclusive. In case of acute and chronic liver allograft rejection, histology remains the gold standard investigation. Acute rejection occurs in 10 to 50 days after the hematopoietic stem cell transplantation. Chronic rejection occurs usually more than 100

days after the transplantation. Sarcoidosis is a systemic non caseous granulomatous disease. Liver is a common location but patient is usually asymptomatic. Evidence based guidelines for this location, treatment guidelines are lacking and the effect of corticosteroids may be inadequate.

Hepatic morphology may be strikingly affected by circulatory disorders. It is usually asymptomatic and is manifested by nonspecific biochemical disturbances and so it remains undiagnosed in many cases. It can be due to impaired blood inflow due to portal vein obstruction, intra or extrahepatic thrombosis, cirrhosis and sinusoidal occlusion. Hepatic vein outflow obstruction can occur due to hepatic vein obstruction (Budd-Chiari syndrome), sinusoidal obstructive syndrome leading to elevated aminotransferases and jaundice.

Chronic liver diseases (CLD) cause significant morbidity and mortality worldwide. Multiple etiological factors lead to a similar clinico-pathological syndrome in CLDs but the rates of progression and clinical course may be different

Liver cirrhosis has become one of the major causes of morbidity and mortality. Hepatitis B virus(HBV) and hepatitis C virus (HCV) are the two major causes of liver cirrhosis. Liver cirrhosis is the end-stage of different chronic liver disease and is often neglected until complications, such as variceal haemorrhage, spontaneous bacterial peritonitis, ascites, or hepatic encephalopathy occur.

Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury that leads to portal hypertension[3].

Before doing liver biopsy, patients should be educated about their liver disease and about investigations other than liver biopsy (if any) that may also provide diagnostic and prognostic information.

AIMS AND OBJECTIVES

To study histopathological spectrum of non-neoplastic liver diseases.

REVIEW OF LITERATURE

Histological assessment of the liver, and thus, liver biopsy, is a cornerstone in the evaluation and management of patients with liver pathology for medical diseases like chronic liver disease. Types of liver biopsies include percutaneous needle core biopsy, fine needle aspiration biopsy done under imaging guidance, trans jugular biopsy and wedge biopsy done during laparoscopic surgery.

Liver biopsy is indicated for diagnosis of multiple parenchymal liver diseases, abnormal liver tests of unknown etiology, fever of unknown origin, focal or diffuse abnormalities on imaging studies, for assessment of prognosis (disease staging), and to assist in making therapeutic management decisions. Liver histology, clinical and laboratory data are used in conjugation to make the diagnosis.

Liver biopsies are divided on the basis of etiology. First category is metabolic causes that include nonalcoholic fatty liver disease, Wilson disease, Gilbert syndrome, Dubin Johnson syndrome and Benign recurrent intermittent cholangitis (BRIC) and Progressive familial intrahepatic cholestasis (PFIC). Second category is drug and toxin induced hepatotoxicity which includes alcoholic liver disease and therapeutic agents including complementary medicines and drugs of abuse. Third category is immune mediated causes like autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, overlap syndrome and transplant rejection. Fourth category is infectious etiology under which comes viral hepatitis caused by hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV) and hepatitis E virus (HEV). Another infectious cause is tuberculosis. Fifth category is mechanical causes that include obstructive cholestasis and vascular disorders like Budd Chiari syndrome. Sixth category is infiltrative disorders like amyloidosis and sarcoidosis. Hematoxylin and eosin stain is used to stain liver biopsies. Special stains usedare Masson's's Trichrome, Reticulin, Perl's, Orcein, Rhodanine, Zeil Nelson and PAS.

NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

It refers to accumulation of excess fat in the liver which is unrelated to consumption of alcohol. It is diagnosed when liver has ≥5% hepatic steatosis without ballooning of the hepatocytes or fibrosis on liver biopsy. NAFLD is a spectrum that ranges from simple steatosis, steatohepatitis with or without fibrosis, cirrhosis and ultimately cirrhosis with HCC. Other causes of fat in the liver like drugs, lipodystrophy, Wilson disease, parenteral nutrition, starvation and viral disease also lead to NAFLD[4]. It is often diagnosed incidentally on imaging and is asymptomatic in majority of the patients and is currently considered as hepatic manifestation of metabolic syndrome 1. The risk of cirrhosis and liver failure is considered minimal during follow-up. Non-alcoholic steatohepatitis (NASH) is diagnosed when liver has ≥5% hepatic steatosis with inflammation and ballooning of hepatocyte which indicate liver injury with or without fibrosis. NASH can progress to advanced fibrosis, cirrhosis and its complication like HCC. NASH cirrhosis is diagnosed in a patient with current or previous histological evidence of steatosis or steatohepatitis.

Spectrum of NAFLD

Simple steatosis- Insulin resistance and its interaction with adipose tissue and associated metabolic syndrome are considered as a key step in the pathogenesis of hepatic steatosis. Insulin resistance leads to dysregulation of many hepatic transcription factors and nuclear receptors, leads to alteration of lipid and glucose metabolism which in turn result in fatty liver. There was a study conducted by Singh et al on patients who were biopsy-proven NAFLD (150 with NAFLD and 261 with NASH)[5]. Patients with NAFLD and no fibrosis at baseline progressed to stage 1 of fibrosis over 14.3 years whereas patients with NASH and no fibrosis at baseline had accelerated rate of progression to stage 1 of fibrosis over 7.1 years. Thus, NAFLD without fibrosis has slow rate of progression to advanced stage of cirrhosis.

Non-Alcoholic Steatohepatitis (NASH) - it is a histological diagnosis and requires liver biopsy for its diagnosis. Presence of steatosis, lobular inflammation and hepatocyte ballooning is the histological

feature that distinguishes NASH from simple steatosis. Its risk factors and progression to fibrosis and cirrhosis have been evaluated in many studies. NAFLD advanced to NASH in 23% of cases over a period of 3 years based on histological diagnosis and follow up biopsies of 52 patients in a study by Wong et al[6]. Risk factors which increase the likelihood of NASH in patients with NAFLD include obesity, older age, female sex, non-African American race, diabetes mellitus, and hypertension[7].

Advanced fibrosis and cirrhosis - Long-term outcomes studies on natural history of advanced fibrosis and cirrhosis due to NAFLD are limited. In a meta-analysis the global incidence of advanced fibrosis in NASH patients was 67.9 in 1,000 person-years, with 41% of NASH patients experienced progression of fibrosis (average annual progression rate of 0.09%). NAFLD patients progressed to cirrhosis in 25% of patients and 7% to end-stage liver disease[8]. Study comparing patients of chronic liver disease due to viral hepatitis patients and HCC and patients with HCC due to NASH related cirrhosis tend to be older and are likely to be females[9]. HCC in NAFLD patients has been associated with increasing age, obesity and diabetes.

Clinical presentation

Most of the patients with NAFLD are asymptomatic or have non-specific symptoms before the diagnosis is made. Fatigue and right upper abdominal discomfort are common presenting symptoms. Other symptoms include abdominal bloating, irregular bowel habits and decreased sleep. NAFLD is incidentally diagnosed in majority of the patients with abnormal liver enzymes or abnormalities on imaging performed for other reasons or are identified based on clinical features of the metabolic syndrome[10]. It has been seen in imaging that 70–80% of subjects with central obesity and 50–80% of patients with type 2 diabetes develop the disease[8,9]. NAFLD has also been reported in patients with dyslipidaemia, obstructive sleep apnoea syndrome, polycystic ovarian disease, hypopituitarism, vitamin D deficiency, hypothyroidism and hyperuricemia. These factors should also be assessed simultaneously while ordering the blood tests for the

evaluation of NAFLD[11,12]. Most of the patients with NAFLD have normal or near normal LFTs. Patients may show mildly raised transaminases (ALT > AST) and/or GGT and even if elevated, the ALT typically falls (and AST may rise) as fibrosis progresses to cirrhosis. ALT level correlation with histological findings has poor sensitivity and specificity for the diagnosis of NASH[13,14]. Patients with abnormal LFTs, should be simultaneously evaluated for alcohol abuse, drug-induced liver injury, viral hepatitis, autoimmune liver disease, haemochromatosis, celiac disease and Wilson's disease (in patients <45 years old). Autoantibodies may be detected in low titre in subjects with NAFLD [antinuclear antibody (ANA) <1:100 and/ or anti-smooth muscle antibody (ASMA) <1:40] but these patients are associated with normal IgG levels. Few patients may have high ferritin levels and usually reflect underlying inflammatory activity or insulin resistance[15]. Liver biopsy should be considered if there is any doubt in diagnosis.

Histomorphology of NAFLD and NASH

Steatosis- Fat accumulation in hepatocytes. It includes 2 types:
a) Microvesicular steatosis- When multiple tiny drops of fat cannot displace nucleus and it remains in the center.
b) Macrovesicular steatosis- When single large droplet of fat displaces the nucleus towards periphery.

Lobular and portal inflammation- Mild mixed inflammatory infiltrates composed of lymphocytes, eosinophils, neutrophils and Kupffer cells. Portal inflammation is absent or mild and is predominantly chronic.

Ballooning- Hallmark of steatohepatitis.

Fibrosis- Persinusoidal and pericellular fibrosis is hallmark of NASH and usually begins in zone 3. It can be highlighted on Masson's trichrome or reticulin staining. With progression of NASH bridging fibrosis, periportal fibrosis and liver cirrhosis may occur. Burn-out NASH can occur in advanced fibrosis with disappearance of necro-inflammatory reactions and steatosis. Cirrhosis related to NASH is usually macronodular or mixed.

Glycogenated nuclei- These are vacuolated nuclei which are commonly seen in NAFLD.

Mallory-denk bodies- Granular, eosinophilic irregular shaped aggregates found in hepatocytes. They are specific for NASH.

Iron deposition-Usually mild and within hepatocytes or sinusoidal lining of reticuloendothelial cells.

Megamitochondria-intrahepatic crystal or round shaped eosinophilic structures.

Apoptotic hepatocytes- round, deeply eosinophilic bodies with or without hyperchromatic nuclear fragments.

NASH Clinical Research Network system for Grading

	Grade
Steatosis	Grade 0 (<5%)
	Grade 1 (5-33%)
	Grade 2 (34-66%)
	Grade 3 (>66%)
Ballooning degeneration	Grade 0 (none)
	Grade 1(mild)
	Grade 2 (moderate to marked)
Lobular inflammation	Grade 0 (none)
	Grade 1 (<2 foci/x20 field)
	Grade 2 (2-4 foci/x20 field)
	Grade 3 (>4 foci/x20 field)

NASH Clinical Research Network system for Staging

Stage	Histological Description
0	No fibrosis
1a	Zone 3 perisinusoidal fibrosis, requires trichrome stain
1b	Zone 3 perisinusoidal fibrosis, seen on H&E
1c	Portal/periportal fibrosis
2	Zone 3+ portal/periportal fibrosis
3	As above with bridging fibrosis
4	Cirrhosis

WILSON'S DISEASE is autoimmune disease. Incidence rate is 1 per 30,000 live births and can present in combination with neuropsychiatry conditions. Pathogenesis is failure to incorporate copper into ceruloplasmin due to mutation in gene ATP7B. Due to this there is defective biliary excretion of copper which accumulates within liver cells exceeding the capacity for ceruloplasmin leading to hepatic injury due to hepatotoxic nature. Biochemical tests include reduced serum ceruloplasmin levels, raised serum copper levels and urinary copper excretion >50ug/24hrs. Liver biopsy mimics that of chronic liver disease. Special stains like orcein and Rhodanine help to detect copper deposition which could be focal. In formalin fixed liver biopsy >250 ug/g dry weight is also helpful for the diagnosis. Copper can also get deposited in putamen of brain and in cornea of eyes leading to Kayser-Fleischer rings.

GILBERT'S SYNDROME is named after Augustin Gilbert (1858-1927), a French physician[16]. It is defined as benign, familial mild unconjugated hyperbilirubinemia which is not due to haemolysis. Routine tests of hepatic function and liver histology are normal. It affects some 2-5% population[17]. Patients with Gilbert's syndrome have a deficiency in bilirubin UGT activity – about 30% of normal. It is inherited as autosomal recessive disease. The condition may be diagnosed incidentally at a routine medical examination or when blood being examined for another reason, for instance, viral hepatitis. Jaundice is mild and intermittent. Bilirubin levels are most often<3 mg/dl and predominantly unconjugated. The liver biopsy appears normal except for mild non-specific accumulation of lipofuscin pigment in the centrilobular zones.

DUBIN JOHNSON SYNDROME (DJS) is a rare autosomal recessive disorder characterised by chronic or intermittent conjugated hyperbilirubinemia. There is absence of functional Multidrug Resistance-associated Protein 2 (MRP2) which is encoded by ABCC2 gene. The MRP2 protein is located in the canalicular membrane of hepatocytes and also at the apical membranes of cholangiocytes and enterocytes. Mutation in ABCC2 gene results in impaired secretion of

conjugated bilirubin and other anionic conjugates into bile. Patients usually present with recurrent episodes of jaundice precipitated by infection, pregnancy, oral contraceptives or drugs. The liver biopsy shows enlarged hepatocytes with coarse brown pigments in the cytoplasm. Hepatocytes stain positive with melanin resulting in an insoluble black precipitate and with Periodic acid-Schiff stain (PAS) depicting brown granular pigments from glycogen, mucin and basement membrane.

BENIGN RECURRENT INTRAHEPATIC CHOLESTASIS (BRIC) AND PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS (PFIC) are a rare hereditary disorders characterized by recurrent and intermittent episodes of cholestasis, episodes of pruritus and jaundice with normal extra hepatic biliary tree. The disease may start in early infancy or childhood. PFIC has three subtypes and progress to cirrhosis in teenage years. Histologically, both are characterized by simple or bland bilirubin stasis with bile deposition in hepatocytes leading to feathery degeneration, canaliculi and Kupffer cells during episodes of cholestasis. In BRIC no evidence of fibrosis is seen as the disease doesn't progress to cirrhosis.

ALCOHOLIC LIVER DISEASE- The diagnosis of ALD is made based on reliable history of significant alcohol intake, clinical examination and laboratory features suggestive of significant alcohol intake. Definition of one standard alcoholic drink is any alcoholic beverage that contains 14 g of alcohol. In men consuming more than 4 standard drinks in any single day is considered significant alcohol intake, similarly in women more than 3 standard drinks in any single day (or more than 7 drinks per week) is considered significant to cause alcohol-related problems[7]. Usual clinical presentation is positive history of alcohol intake with fatty liver on ultrasonography. Liver enzymes are not very useful for the diagnosis of patients with ALD as liver enzymes may or may not be elevated as in case of cirrhosis and the elevation does not correlate with severity of disease. However, the pattern of elevation is helpful in making a diagnosis of liver injury. AST is typically two to three times greater than ALT in these patients. Majority of times the levels of AST

and ALT do not increase >5 times the upper limit of normal. Elevated serum gamma-glutamyl transpeptidase (GGT) is also a clue to the diagnosis which is more than serum alkaline phosphatase level.

ALD is a broad spectrum of disease and it can range from totally asymptomatic disease (as fatty liver) to AH and advanced ALD which includes complications of cirrhosis like jaundice, ascites, variceal bleeding, hepatic encephalopathy, and hepatocellular carcinoma (HCC). A study was conducted by Julien et al which aimed to project trends in alcohol-related cirrhosis and deaths in the USA up to 2040[18]. Study has concluded that age-standardised deaths due to alcohol-related liver disease are expected to increase from 8·23 per 100 000 person-years in 2019 to 15·20 per 100 000 person-years in 2040. Thus, without substantial changes in drinking culture the disease burden and deaths due to alcohol-related liver disease will worsen in the USA in the coming future.

Fatty liver- it is seen in majority of the patients consuming alcohol. It is asymptomatic in majority of patients or presents with mild right upper quadrant discomfort. Liver function tests (LFTs) are either normal with normal liver enzymes or mildly increased serum aminotransferase levels and GGT. Liver enzymes (AST and ALT) never rise five times of upper normal value in majority of patients. Ultrasound of the abdomen shows steatosis of various grades (grade 1 to 3). Clinical history along with laboratory findings are sufficient to diagnose this condition and liver biopsy is not required in majority of these patients.

Liver biopsy if done shows macro vesicular steatosis predominantly in the centrilobular zone. There will be no significant inflammation or necrosis in fatty liver disease alone[19]. Now a days, the presence of steatosis on liver biopsy is no longer considered a benign condition. In a study by Teli et al, patients with a histologic diagnosis of alcoholic fatty liver without AH were the subjects (n=88) and were analysed retrospectively[20]. Of these patients, 18% progressed to fibrosis or cirrhosis in span of 10 years. Continued alcohol intake, female gender and histological features of megamitochondria and perivenular fibrosis and mixed macro and micro vesicular steatosis on liver biopsy were

associated with development of fibrosis or cirrhosis. Similarly, in a systemic review by Parker et al mortality was 6% per year in patients with steatosis and hence steatosis no longer considered as benign condition[21].

Alcoholic Hepatitis- it is a severe form of liver injury with features of both hepatocellular injury and cholestasis. It results from significant amount of alcohol consumption during a prolonged period of time. Patient with mild AH may be asymptomatic or mildly symptomatic. Clinical examination of abdomen shows hepatomegaly and laboratory tests shows increased serum aminotransferase levels with the AST level twice or greater than the ALT level. Severe disease is associated with various grades of malnutrition, jaundice, fever, malaise, tender hepatomegaly with or without hepatic bruit. In severe AH, patients may present with ascites, hepatic encephalopathy, and acute kidney injury. Liver biopsy shows neutrophils as predominant inflammatory infiltrate, Mallory hyaline, ballooned hepatocytes and varied degree of fibrosis or cirrhosis as overlap of these pathological findings are common. Perisinusoidal chicken-wire fibrosis and perivenular fibrosis is a distinctive feature of steatohepatitis.

In a study by Parkeret al progression of pre-cirrhotic disease to cirrhosis were 1% in patients with normal histology, 3% in patients with HS, 10% in patients with biopsy suggestive of steatohepatitis and 8% (3–19%) in patients with significant fibrosis[21]. Mortality was significantly higher in patients with cirrhosis compared to those with steatosis.

Alcoholic cirrhosis- Alcohol is most common hepatotoxic agent and excess and continued intake of alcohol consumption increases the risk of developing cirrhosis. Alcoholic cirrhosis liver is characterized by small regenerative nodules (micronodular). Absolute abstinence of alcohol significantly improves long-term prognosis and hence should be reinforced in every patient[22].

VIRAL HEPATITIS- It can be of viral etiology or drug induced. Viral hepatitis is acute and chronic.

Acute viral hepatitis is caused by Hepatitis A virus (HAV) and

Hepatitis E virus (HEV). HAV is a benign, self-limited disease with incubation period of 2 to 6 weeks. It is RNA virus and affects children mainly via contaminated water and food. Viremia is transient and IgM antibodies appear with onset of symptoms followed by IgG antibodies. HEV is RNA virus affecting young to middle aged people via contaminated water and food. In acute hepatitis biopsy is seldom taken and diagnosis is based on serology. It is lobule predominant pathology. There is lobular disarray with ballooning degeneration of hepatocytes, spotty or confluent necrosis and acidophilic bodies. Lobular inflammation by lymphoplasmacytic infiltrate is common. Features of regeneration are also evident.

Chronic viral hepatitis include hepatitis B and C.*HCV* is an RNA virus related to Flaviviridae and some RNA plant viruses. It is a 50–60 nm, enveloped, single-stranded, positive-polarity RNA virus. Chronic HCV infection occurs all over the world. It is estimated that globally 110 million people are HCV-antibody positive and 80 million have chronic infection[23]. HCV may augment liver lesions in chronic alcoholism and can also lead to hepatocellular carcinoma. In India approximately 12–18 million people are thought to be infected with HCV[24]. The prevalence of HCV infection in the general population of Punjab was studied by Sood et al. in the year 2012, where a house-to-house survey was conducted in a defined population of 26,273 subjects and 5258 subjects were screened[25]. The prevalence of HCV was 5.2% with the highest prevalence being noted in the age group of 41–60 years and prevalence was no different among men and women. The authors also assessed the risk factors associated with the acquisition of HCV and reported prior history of dental treatment, surgery, and unsafe sexual practices as the leading causes for HCV infection. The prevalence of HCV seropositivity was very high in certain districts of Punjab such as Mansa (13.6%), Muktsar (12.4%), Moga (8.8%), and in Sangrur (5.7%). The gold standard for diagnosis has been the identification of viral RNA using the polymerase chain reaction.

Mode of transmission ofhepatitis Cvirus is via blood borne. It is most commonly transmitted through sharing of injection equipment,

inadequate sterilization of medical equipment, transfusion of unscreened blood and blood products, sexual practices that lead to exposure to blood. Histological features include marked and patchy expansion of portal tracts by predominantly lymphocytic infiltrate with minimal interface hepatitis into adjacent lobule. There is a well-defined lymphoid aggregate, including true follicle formation with a well-defined germinal center. Varying degrees of bile duct damage and even focal bile duct loss can also be seen. Micro-and macrovesicular steatosis is the common feature encountered. Sinusoidal cell hyperplasia can be seen. Less commonly seen features include slight lobular necrosis, liver cell dysplasia, multinucleation and accumulation of Mallory-like material in hepatocytes. These features are not pathognomonic and have been recognized in other forms of chronic hepatitis including hepatitis B, but they are most often seen in hepatitis C.

The mature *HBV* virion is a spherical, double layered “Dane particle” that has an outer envelope of protein, lipid and carbohydrate enclosing an electron dense core. There is a nucleocapsid core protein called HBcAg, envelope glycoprotein called HBsAg. Also present is Polymerase (Pol) that exhibits both DNA polymerase activity and reverse transcriptase activity. HBx protein is necessary for virus replication. The natural course of the disease can be followed by serum markers. HBsAg appears before the onset of symptoms, peaks during overt disease and then declines to undetectable levels in 12 weeks. Anti-HBs antibody arises after acute infection is over and may persist for life. HBeAg, HBV-DNA and DNA polymerase appear in serum soon after HBsAg and all signify active viral infection.

Mode of transmission in 90% cases is horizontal that is during the childbirth. Unprotected sex and intravenous drug abuse are other chief modes of spread of the disease. Histological features include ground glass hepatocytes which are cells with endoplasmin reticulum swollen with HBsAg. Sanded nuclei are seen due to accumulation of HBcAg in hepatocyte nuclei. Cholestasis is a late feature which can be seen. A study was conducted by Cornianu et al[26] in which the frequency of portal lymphoid follicles (LF) and portal lymphoid aggregates (LA)

(68%), and Mallory-like material (MM) was extremely significantly increased in HCV vs. HBV patients (LA/LF in 20%). It was also found that the incidence of bile duct lesions (BDL) is very significantly increased in HCV (44%), in comparison with HBV infection (21%).

The Ishaak modified histological activity index (HAI) grading system is used for chronic viral hepatitis.

Interface hepatitis	Score
Absent	0
Mild	1
Mild/moderate	2
Moderate	3
Severe	4
Confluent necrosis	
Absent	0
Focal confluent	1
Zone 3 necrosis in some	2
Zone 3 necrosis in most	3
Zone 3 necrosis + occasional portal to central bridging	4
Zone 3 necrosis+ multiple portal to central bridging	5
Pan acinar/multinecrosis	6
Focal lytic necrosis	
Absent	0
<1 focus/10x	1
2-4 focus/10x	2
5-10 focus/10x	3
>10 focus/10x	4
Portal inflammation	
None	0
Mild some or all portal tracts	1
Moderate some or all portal tracts	2
Moderate to marked	3
Marked	4

Staging is done on the basis of fibrosis as per Metavir staging system.

Staging	Score
None (0)	0
Enlarged / fibrotic portal tract (1)	1
Periportal /portal-portal fibrosis (2)	2
Fibrosis with architectural distortion but no obvious cirrhosis (3)	3
Probable / definite cirrhosis (4)	4

Autoimmune hepatitis is a chronic progressive hepatitis with all features of autoimmune disease in general: genetic predisposition, presence of autoantibodies and therapeutic response to immunosuppression. The diagnosis of autoimmune hepatitis (AIH) is clinico-pathological and is based on a combination of biochemical, immunological and histological features and exclusion of other causes of liver disease. Based on the patterns of circulating antibodies AIH is classified as Type 1 and 2. Antinuclear antibodies (ANA), anti-smooth muscle actin (SMA), anti-soluble liver antigen/liver pancreas antigen (anti SLA/LP) antibodies and antimitochondrial antibodies (AMA) are seen in type 1. Anti-liver kidney microsome 1 (anti LKM-1) are seen in type 2.

Autoimmune hepatitis shows extensive interface hepatitis, plasma cell predominance and hepatocyte rosettes in areas of marked activity.

Liver biopsy is considered a critical element in the diagnosis of autoimmune hepatitis and independent factor to distinguish autoimmune hepatitis from other liver diseases. According to simplified criteria proposed by International Autoimmune Hepatitis Group (2008) definite and probable AIH is given by considering for points: autoantibodies, IgG, liver histology and absence of viral hepatitis. Histologic features to be present in same biopsy to qualify as a histologically ‘typical’ (2 points): interface lymphocytic /

lymphoplasmacytic inflammatory activity, rosettes, and emperipolesis.1 point is compatible with AIH and 0 point atypical for AIH.

Simplified diagnostic criteria (2008) of the International Autoimmune Hepatitis Group

Variable	Cut-off	Points
ANA or ASMA	>1:40	1
ANA or ASMA Or LKM Or SLA	>1:80 >1:40 Positive	2
IgG	>Upper Normal Limit	1
	>1.10 x Upper Normal Limit	2
Liver histology	Compatible with AIH	1
	Typical AIH	2
Absence of viral hepatitis	Yes	2 >6: probable AIH >7: definite AIH

Typical and compatible histology for the diagnosis of AIH in the 2008 simplified criteria

Diagnostic category	Histologic features
Typical AIH 2 points	Interface hepatitis with lymphocytic or lymphoplasmacytic portal inflammatory infiltrates extending into the lobules Hepatocyte rosette formation Emperipolesis
Compatible with AIH 1 point	Chronic hepatitis with lymphocytic infiltration without all features considered typical
Atypical 0 points	Evidence of another diagnosis

The portal inflammatory infiltrate in AIH ranges from mild to severe and includes lymphocytes, histiocytes, plasma cells, eosinophils and neutrophils. Predominance of plasma cells in the portal infiltrate is

typical for AIH but they are present only in 2/3 of biopsies and their absence does not exclude the diagnosis[27]. Usually, a majority of plasma cells in AIH are Ig G-positive by immunohistochemistry, and the IgM/IgG ratio may aid in the differential diagnosis from PBC in cases with significant bile duct injury[28]. Interface hepatitis with spill-over of inflammatory cells into the periportal parenchyma and hepatocyte necrosis is observed in 84–98% of the cases, is more prominent in disease flares, and is usually more severe in AIH compared to chronic hepatitis of other aetiology. Emperipolesis, characterised in AIH by the presence of a lymphocyte or rarely a plasma cell within the cytoplasm of hepatocytes is reportedly found in 65–78% of AIH cases[29, 30]. It occurs more commonly in AIH compared to chronic viral hepatitis, PBC or drug induced liver injury (DILI) and usually entails CD8+ Tlymphocytes. Physiologically, emperipolesis and subsequent degradation of autoreactive Tlymphocytes is important for the maintenance of immunetolerance. Interference with this mechanism may result in the accumulation of viable autoreactiveT-lymphocytes in the liver and breach of tolerance leading to the development of AIH[31]. In AIH, emperipolesis may inducehepatocyte apoptosis and has been proposed as an additional mechanism of autoimmune-mediated hepatocyte injury. AIH patients with emperipolesis in liver biopsy have significantly higher serumtransaminase levels, more severe necroinflammatory activity and more advanced fibrosis[30].

Hepato cellular rosettes is diagnostic histological feature and is an indication of hepato cellular regeneration following lobular injury. Rosettes are small groups of hepatocytes arranged around a small, sometimes not visible, central lumen. The presence and extent of rosette formation is better appreciated on reticulin stain. Rosetting is more common in AIH (49% of cases) compared to chronic viral hepatitis. In paediatric patients, rosette formation has emerged as the most significant histologic predictor of AIH in biopsies with chronic hepatitis.

PRIMARY BILIARY CIRRHOSIS (PBC) is a slowly progressive liver disease. It has unknown aetiology and affects mainly middle-aged

women. The serum bilirubin concentration is the best laboratory test with prognostic relevance. The liver enzyme pattern is cholestatic, in that ALP and g-glutamyl transferase (g-GT) levels are elevated. Also, the concentration of IgM is elevated in almost all patients. Bilirubin rises in the end stage of the disease. The diagnosis of PBC is based on three criteria: presence of detectable anti-mitochondrial antibodies (AMA) in serum; elevation of liver enzymes (most commonly alkaline phosphatase (ALP)) for >6 months; and histological findings in the liver that are compatible with the presence of the disease. A probable diagnosis requires the presence of two criteria and a definite diagnosis requires all three. Antimitochondrial antibodies are the hallmark of PBC. Antinuclear antibodies (ANA) may also be found. In a study conducted by Drebber et al, AMA positivity was seen in 84% cases and ANA positivity in 61% cases[30].

Histologically, the interlobular bile ducts are affected, causing chronic non suppurative destructive cholangitis. The typical diagnostic lesion of PBC is the florid duct lesion with formation of non caseating epithelioid granulomas. The cholangitic process results in loss of these bile ducts, associated with ductularreaction, periportal hepatitis and liver cirrhosis. Drebber et al also concluded that tentative diagnosis of PBC could be made in the basis of histologic features in 61% cases[32].

Histological staging is an important part of clinical studies currently carried out in many centres and is used routinely in some of the schemes applied for predicting survival following liver transplantation.

PRIMARY SCLEROSING CHOLANGITIS- it is a chronic cholestatic disorder characterised by nonspecific inflammatory fibrosis in the wall of the biliary tree leading to stenosis and ectasia. It affects infants and children with male preponderance. It has strong association with inflammatory bowel disease. Imaging of the biliary tree is extremely important which demonstrate irregular areas of stricture and beading of the bile ducts, in 80% of the patients and involves intra and extrahepatic bile ducts. Pathological features include onion skin type of periductal fibrosis around medium sized or large ducts with

degeneration and atrophy of the epithelial lining and replacement of bile duct by fibrous cords. Ductopenia can be seen.

Overlap syndrome is the term used for patients presenting with features of various autoimmune liver diseases (i.e., AIH, PBC and PSC). Overlap syndrome lacks specific definitions. The AIH-PBC overlap syndrome is accepted when two or three criteria for PBC and AIH are fulfilled. For AIH - the criteria are serum IgG levels two times the upper limit of normal (ULN), strongly positive for ANA and liver biopsy showing interface hepatitis. For PBC, the criteria are serum tests strongly positive for AMA-M2 and nuclear membrane and liver biopsy showing bile duct lesions. PBC in genetically predisposed patients of AIH can flare up autoimmune destruction of bile ducts which results in mixed clinical presentation of AIH and PBC in such patients[33].

PBC enhances the autoimmune mechanism and genetic predisposition along with resulting inflammatory hepatitis with most of the features of AIH

DRUG-INDUCED LIVER INJURY (DILI) can mimic any pattern of primary liver disease. A thorough clinical history, including exposure to herbal, over-the-counter agents and toxins, along with a systematic literature search, is critical to establish the diagnosis. Acute hepatitis, with or without cholestasis, is the most common histological pattern of drug-induced liver injury. Drugs such as acetaminophen is the most common cause of acute liver failure in the USA. Drug-induced chronic hepatitis is rare, but fibrosis and cirrhosis can occur with drugs such as methotrexate, while autoimmune hepatitis-like disease can result with drugs such as minocycline. In some instances, drug-related cholestatic injury can be prolonged and can lead to ductopenia. Drug-induced steatohepatitis is a rare phenomenon, but is well known to occur with amiodarone and irinotecan. Many drugs, such as tamoxifen, oestrogenic drugs and nifedipine, can precipitate or exacerbate steatohepatitis in the presence of other risk factors.

Drugs may cause liver injury in a predictable dose dependent manner in most human and animal settings (intrinsic DILI) or in an unpredictable, non–dose-dependent manner (idiosyncratic DILI). In India there is high incidence of ayurvedic and herbal medicine intake. A study was conducted in South India by Philips et al in which it was found that ayurvedic and herbal medicines lead to severe liver injury[34], Ayurveda and herbal products are implicated as a cause of hepatotoxicity in 2% to 11% of patients and India, showing a prevalence of 1.3%[35].

List of hepatotoxic drugs

LESION	EXAMPLES OF COMMONLY USED DRUGS
Intrinsic hepatotoxicity	
Microvesicular steatosis	Valproate
Phospholipidosis	Amiodarone
Hepatocellular necrosis/acute liver injury	Paracetamol
Fibrosis	Vitamin A
Cholestasis	Contraceptive steroids
Idiosyncratic hepatotoxicity	
Hepatitis	Isoniazid
Cholestasis	Amoxicillin-clavulanic acid
Granuloma formation	Allopurinol

Pathological patterns are acute hepatitic, chronic hepatitic, acute cholestatic, chronic cholestatic, cholestatic-hepatitic, granulomatous, macrovesicular steatotic, microvesicular steatotic, steatohepatitic, zonalnecrosis, non-zonal necrosis, vascular injury, hepatocellular alteration, nodular regenerative hyperplasia, mixed or unclassified injury, minimal nonspecific changes, absolutely normal, and massive necrosis.

Acute Hepatitis-DILI accounts for 10% of acute hepatitis and is the most common cause of cholestatic hepatitis. A wide variety of drugs

can cause acute hepatocellular injury. Herbal and botanical drugs are an important cause of hepatotoxicity. The morphological patterns can be observed in acute hepatocellular injury are acute hepatitis, necrosis and resolving hepatitis.

Acute Liver Failure (ALF) is defined as the onset of hepatic encephalopathy within 8 weeks of onset of symptoms. Drugs are the most common cause of ALF in the USA, accounting for 25–50% of cases. Morphological features in ALF are extensive microvesicular steatosis and necrosis with marked inflammatory activity.

Chronic Liver Disease typically refers to persistent biochemical abnormalities beyond 6 months. In some series, the cut-off of 3 months has been used for hepatocellular injury and 6 months for cholestatic or mixed injury. Progression to chronicity has been reported in 5–10% of adverse drug reactions.

Acute Cholestatic Injury-Drug-induced cholestatic injury can manifest clinically with jaundice, pruritus, dark urine and pale stools. Liver enzyme studies typically reveal elevation of alkaline phosphatase and glutamyl transferase. Histologically it can present as bland cholestasis or cholestatic hepatitis. Antibiotics like amoxicillin-clavulanic acid cause this pattern of injury.

Chronic Cholestasis and Ductopenia- Drugs causing prolonged cholestasis (defined as greater than 3 months in duration) and ductopenia include antibiotics such as amoxicillin–clavulanic acid and flucloxacillin, antifungals such as terbinafine and, rarely, oral contraceptives. Amiodarone can also cause prolonged disease.

Granulomatous Hepatitis- The most common causes of granulomas in the liver are infections, sarcoidosis, primary biliary cirrhosis and drugs. Granulomas are uncommon in hepatitis C but can occur in patients treated with interferon. Talc granulomas can occur in intravenous drug users and can be detected by viewing under polarised light.

Steatosis and Steatohepatitis- Macrovesicular steatosis includes large and small droplet fat. The term ‘‘large droplet fat’’ is used when at least half the hepatocyte cytoplasm is occupied by a single lipid vacuole, while multiple lipid vacuoles are seen in small droplet fat. It usually

occurs as a side effect of steroids, nitrofurantoin, gold, methotrexate, NSAIDs such as ibuprofen. Microvesicular steatosis is result of mitochondrial injury and often occurs as an adverse effect of drugs/toxins such as cocaine, tetracycline, valproic acid and zidovudine. Steatohepatitis is characterised by steatosis, lobular inflammation and hepatocellular injury in the form of hepatocellular ballooning (with or without acidophil bodies or Mallory hyaline) or pericellular fibrosis. A few drugs (notably amiodarone and irinotecan) play a direct aetiological role in steatohepatitis.

Vascular Abnormalities- it includes Sinusoidal obstruction syndrome, Peliosis hepatis and Hepatic vein thrombosis.

AMYLOIDOSIS refers to spectrum of diseases resulting from extracellular deposition of fibrillar amyloid protein. M:F ratio is 2:1 and usually affects 6^{th} to 7^{th} decade of life. 9% of patients with amyloidosis have liver involvement and more than 30% of patients with systemic amyloidosis have clinical evidence of hepatic involvement[36]. Autopsy studies have reported 56-95% of patients with systemic amyloidosis with hepatic involvement[37]. Hepatomegaly has been reported in about 81-92% patients with hepatic amyloidosis but this does not necessarily indicate the presence of Primary Hepatic Amyloidosis (PHA),especially when patients are complicated with congestive heart failure.

It is divided into five types: primary amyloidosis, secondary amyloidosis, familial amyloidosis, amyloidosis of aging (or senile amyloidosis), and dialysis related amyloidosis. Accordingly, systemic amyloidosis has been recently classified as AL, AA, Apo A1, ATTR, and other types. Protein misfolding, aggregation, and fibril formation are important mechanisms for amyloidosis. Primary hepatic amyloidosis refers to abnormal deposition of the AL amyloid materials in the liver.

Macroscopically, the liver may look waxy grey with a smooth surface, blunt edge, moderate hardness, and poor elasticity. In advanced cases with massive amyloid infiltration, the liver may swell and become rubbery in consistency, giving a “lardaceous liver” appearance on cut surface. Splenomegaly is often present as a result of portal

hypertension. Microscopically, amyloid deposits are usually found in the hepatic parenchyma, along the sinusoid within the space of Disse, or in vessel walls. Hepatocytes are severely compressed by extensive accumulation of amyloid, leading to cellular atrophy and diminished cell number. Congo Red is regarded as the gold standard stain.

Hepatic granulomas can be caused by variety of disorders as shown in table. Some of these are primary in the liver but most are part of a generalized disease process.

GRANULOMATOUS DISEASES

Etiology of Hepatic granulomas

Aetiology	Favoured site	Special features
Sarcoidosis	Portal/periportal	Clustering Hyalinisation inclusions in giant cells May destroy bile ducts
Tuberculosis	None	Necrosis
PBC	Portal	Near damaged bile ducts
Drug	None	Eosinophils Other lesions present(hepatitis, fat, cholestasis)
Mineral oil	Portal, perivenous	Oil vacuoles
Q fever, CMV	None	Fibrin ring granulomas
Ch. Granulomatous ds. Of childhood	None	Brown pigment in macrophages
Cat scratch disease, tularemia	None	Purulent centre

HEPATIC TUBERCULOSIS is common and has a worldwide distribution. It is diagnosed clinically in 50-80 percent of all patients dying of pulmonary tuberculosis and in up to 91 percent at autopsy[38]. Hepatic tuberculosis can be classified into miliary and localized forms.

The former is associated with miliary dissemination which occurs from hematogenous spreading via the hepatic artery. In the local form, bacteria reach the liver from the intestine via the portal vein. Both forms of hepatic tuberculosis have been seen in normal and immunocompromised hosts. Biochemical investigations include raised alkaline phosphatase levels and reversal of A/G ratio. Imaging shows hepatomegaly, diffusely increased parenchymal echogenicity to multiple hypoechoic lesions in the liver. On microscopy there are caseating granulomas in the hepatic parenchyma. But this is a nonspecific finding seen in many conditions. PCR is a valuable tool for the demonstration of mycobacterial DNA in tissues which may be more reliable than histopathology for detecting *Mycobacterium tuberculosis* in a liver biopsy.

A study conducted by Drebber et al showed that 442 liver biopsies were of granulomatous hepatitis and etiologies were PBC in 48% cases, 8% had sarcoidosis and 2% were due to drugs[39].

SARCOIDOSIS is a multisystemic inflammatory granulomatous disease of unknown aetiology. Typical for sarcoidosis is the presence of non-necrotizing granulomas, affecting lung and lymph nodes in more than 90% of the cases, but it can involve almost every organ system. Extrapulmonary sites, including the liver, are involved in 50% of the cases[40]. Hepatic sarcoidosis mostly affects young individuals between 20 and 40 years old, and is twice as common in African-Americans as in Caucasians. Patients present with atypical clinical signs and symptoms such as nausea, vomiting, abdominal pain in the right upper quadrant, a painful liver during palpation and/or hepatomegaly. In a minority of patients, hepaticsarcoidosis can be a serious and rapid progressive disease with the occurrence of complications as cirrhosis, portal hypertension, chronic cholestasis and Budd–Chiari syndrome. Diagnosis of hepatic sarcoidosis is established when clinico-radiological findings are supported by a compatible histological picture of a biopsy obtained from another organ and/or the liver, after the exclusion of other causes of hepatic granulomas. On microscopy tiny

naked granulomas are seen. Necrosis is usually absent. Schaumann's bodies and asteroid bodies can be noted.

Hemosiderosis is iron accumulation in Kupffer cells and macrophages due to any cause.

In hemochromatosis there is deposition of golden yellow hemosiderin granules in cytoplasm of periportal hepatocytes due to underlying genetic cause. Perl's Prussian blue stain helps to interpret liver biopsy. It is given as primary or secondary then gradation from periportal to center. Also look for iron free or iron less zones as these may harbor dysplastic changes.

Grading for iron on Perl's stain

Grade 0	Iron granules absent
Grade 1	Iron granules seen at 250x
Grade 2	Iron granules seen at 100x
Grade 3	Iron granules seen at 25x
Grade 4	Iron granules seen at 10x

Hereditary defects of bilirubin metabolism lead to unconjugated and conjugated hyperbilirubinemia. Unconjugated bilirubin is raised in Crigler Najjar syndrome type I and II and Gilbert syndrome. These syndromes are differentiated on the basis of UGT1A1 residual enzymatic activity and its affected bilirubin levels and responsiveness to Phenobarbital treatment. Dubin Johnson syndrome leads to conjugated hyperbilirubinemia due to mutation in canalicular multidrug resistance protein (MRP 2). Liver biopsy shows darkly pigmented cytoplasmic globules.

Among the outflow vascular pathology is Budd-Chiari syndrome. The obstruction may be thrombotic or non-thrombotic anywhere along the venous course from hepatic venules to junction of inferior vena cava.

LIVER TRANSPLANTATION- it has been accepted as the main therapeutic option for patients with end-stage liver diseases. Liver histology remains the "gold standard" for the diagnosis of allograft dysfunction. Histopathology helps to examine the host liver to be

removed at transplantation. In transplanted liver, in acute rejection there is infiltration by mixed portal inflammatory infiltrates with bile duct injury and endothelitis. In chronic rejection there is ischemia of liver parenchyma due to obliteration of small and large arteries. Destruction of bile ducts leads to vanishing bile duct syndrome requiring re-transplantation.

In a study done in Iran, a total of 382 needle biopsies were performed on 287 patients aged between 1 and 64 years old. Acute rejection was the most common diagnosis, which occurred in 180 (47%) of specimens. Among other complications were vascular problems (8.6%), preservation/reperfusion (I/R) injury (7%), chronic rejection (5.2%), biliary injury/obstruction (3.4%), recurrence of primary disease (2.6%), drug-induced hepatic injury (1.8%), cirrhosis (1.6%), sepsis (1.4%), cytomegalovirus hepatitis (1.4%), post-transplantation lymphoproliferative disease (1%) and Venous outflow obstruction (0.5%)[41].

So**,** the most common pathological diagnosis of post-transplant liver needle biopsies has been acute rejection, followed by ischemia due to hepatic artery thrombosis, preservation/reperfusion injury, and chronic rejection.

Banff scheme for grading liver allograft rejection- Rejection activity index

Category	Score
Portal inflammation	3
Bile duct inflammation/ damage	1
Venular endothelial inflammation	3

LIVER CIRRHOSIS is the final pathological result of various chronic liver diseases, and fibrosis is the precursor of cirrhosis. Many types of cells, cytokines and miRNAs are involved in the initiation and progression of liver fibrosis and cirrhosis. Activation of hepatic stellate cells (HSCs) leads to fibrosis. Activated Kupffer cells destroy hepatocytes and stimulate the activation of HSCs. Repeated cycles of

apoptosis and regeneration of hepatocytes contribute to pathogenesis of cirrhosis. At the molecular level, many cytokines are involved in mediation of signalling pathways that regulate activation of HSCs and fibrogenesis. Recently, miRNAs as a post-transcriptional regulator has been found to play a key role in fibrosis and cirrhosis[42]. The data of this study confirm that CC likely results from the progression of unrecognised NASH in a large proportion of cases; hence it maybe better defined as “metabolic cirrhosis” in most cases. The subset of lean patients with Cryptogenic Cirrhosis, however, may suggest non-metabolic risk factors, and this group therefore deserves further investigation. The predictors of hepatic complications and death in patients with CC are similar to those of cirrhosis due to HCV. Patients with CC have a high risk of developing severe liver complications and a poor prognosis owing to liver-related death and cardiovascular and oncologic co-morbidities. The risk of HCC developing in CC, even in the early phase, is not negligible, and thus warrants the setting up of surveillance programs for early detection of NAFLD-NASH in patients with dysmetabolic features[43].

Cirrhosis and other chronic liver diseases are a major cause of morbidity and mortality globally. Burden and underlying causes differ across locations and demographic groups. A study was conducted on Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) 2017 on the burden of cirrhosis and its trends since 1990.Basis of the study were cause, sex, and age, for 195 countries and territories. Cirrhosis imposes a substantial health burden on many countries and this burden has increased at the global level since 1990, partly due to population growth and ageing. Although the age standardised death and Disability adjusted Life years (DALY) rates of cirrhosis decreased from 1990 to 2017, numbers of deaths and DALYs and the proportion of all global deaths due to cirrhosis increased. Despite the availability of effective interventions for the prevention and treatment of hepatitis B and C, they were still the main causes of cirrhosis burden worldwide, particularly in low-income countries. The impact of hepatitis B and C is expected to be attenuated and overtaken by that of NASH in the near

future. Cost effective interventions are required to continue the prevention and treatment of viral hepatitis, and to achieve early diagnosis and prevention of cirrhosis due to alcohol-related liver disease and NASH[44].

MATERIAL & METHODS

SOURCE OF DATA

The present 5 years {1.5 year prospective analysis (January 2019 – June 2020) and 3.5 year retrospective analysis (July 2015 – December 2018)} study of trucut liver biopsies was conducted in the Department of Pathology, Dayanand Medical College and Hospital, Ludhiana. During this period a total of 364 cases were analysed. The clinical, biochemical and radiological findings were recorded according to the proforma.

METHOD OF COLLECTION OF DATA

INCLUSION CRITERIA: All the trucut and wedge liver biopsies of non-neoplastic liver diseases.

EXCLUSION CRITERIA: Liver resections, explants specimen or biopsies with neoplastic etiologies.

METHODOLOGY: Liver biopsies were received in container containing 10% formalin. Tissue was processed and stained with heamtoxylin and eosin (H&E) stain. Special stains like masson's trichome, reticulin, Periodic Acid Schiff (PAS), prussian blue, rodanine, orcien were done wherever required. All the biopsy specimen received were correlated with liver function tests which include bilirubin, AST, ALT, GGT, albumin and prothrombin time.

DURATION OF STUDY: Retrospective 3 ½ years and prospective 1 ½ years.

STUDY DESIGN: This is prospective and retrospective study.

OUTCOME: All the findings of specimen will be documented according to the protocol.

STATISTICAL ANALYSIS: Data was described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations were done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

PARAFFIN BLOCK TISSUE PROCESSING:

The tissue fixed in 10% formalin was processed through a series of solutions in the automatic tissue processor as follows:

- Acid formalin for 4 hours
- Formal alcohol for 1 hour
- 80% ethyl alcohol for 1 hour
- 90% ethyl alcohol for 1 hour
- Absolute ethyl alcohol for 1 hour
- Acetone (1st change) for 1 hour
- Acetone (2nd change) for 1 hour
- Xylene (1st change) for 1 hour
- Xylene (2nd change) for 1 hour
- Paraffin (1st change at 58-60°C) for 1 hour
- Paraffin (2nd change at 58-69°C) for 1 hour

The paraffin embedded tissue blocks were cut at 2-3 microns thickness by rotary microtome. These sections were then transferred onto albuminized slides. These sections were labelled properly with the help of glass marking pencil.

PROCEDURE OF HEMATOXYLIN AND EOSIN STAINING

1. Slides were kept on hot plate in order to dewax the sections at temperature up to 60°C.
2. Then slides were deparaffinised in xylene.
3. Then hydration was done by passing through descending rates of alcohol to water.
4. The slides were then stained in Harris Hematoxylin for 15 minutes. Sections were washed well in running tap water until sections turn blue. These sections were then differentiated in 1% acid alcohol (1% HCI in 70% alcohol) for 5-10 seconds, washed well in tap water till sections were again blue for 5 minutes or less.
5. These were again stained in 1% Eosin for 10 min followed by washing in running tap water for 1-5 minutes.

6. Sections were dehydrated once again through ascending grades of alcohol, cleaned in Xylene and mounted with DPX (distyrenedibutylphtalate xylene) avoiding air bubbles.

7. Stained slides were then subjected to microscopic examination under both low (10x) and high power (40x).

PROCEDURE FOR MASSON'S TRICHROME STAIN

1. The slides were washed under water.
2. Mercury pigment was removed by iodine (iodine bleach with sodium thiosulphate for 5 minute)
3. Slides were washed in tap water again
4. Nuclei were stained by Celestin blue haematoxylin for 5 min
5. Differentiated with 1% acid alcohol
6. Washed in tap water
7. Stain with acid fuschin for 5 minute
8. Rinsed in water
9. Treated with phosphomolybdic acid for 5 minutes.
10. Slides were then drained.
11. Slides were then stained with methyl blue for 2-5 minutes.
12. Rinsed in water
13. Treated with 1% acetic acid for 2 minutes
14. Dehydrated through alcohol & clear and then mounted in DPX.

PROCEDURE FOR RETICULIN STAIN

1. Deparaffinize
2. Wash with H_2O
3. 25% Potassium permagnate for 2 minutes
4. Wash well with H_2O
5. Decolorize with 5% oxalic acid until section become clear.
6. Wash with H_2O
7. Add ferric chloride
8. Give wilder bath for 1 minute
9. Wash with H_2O
10. Just dip in formalin (become black)
11. Place gold chloride for 10 minutes
12. Wash with H_2O

13. Place 5% sodium thiosulphate solution for 5 minutes.
14. Wash with water
15. Counter stain with Neutral red.

PROCEDURE FOR RHODANINE

1. Take Test and control sections to water
2. Incubate in the rhodanine working solution at 56 c for 3 hours or overnight in at 37 c oven.
3. Rinse in several changes of distilled water for 3 minutes.
4. Stain in acidified Lillie _ Mayer or other alum hematoxylin for 10 seconds.
5. Briefly rinse in distilled water and place immediately in borax solution for 15 seconds.
6. Rinse well in distilled water.
7. Mount with apathy s mounting media.

PROCEDURE FOR ORCEIN

1. Deparaffinize sections and take to alcohol.
2. Place in staining solution in Coplin jar, 1 – 3 hours at room temperature or 1 hour at 56 c.
3. Rinse in tap water.
4. Counter stain as desired (van Gieson, eosin or trichrome methods are applicable).
5. Dehydrate through ascending grades of alcohol.
6. Clear in Xylene and mount

PROCEDURE FOR PERL'S PRUSSIAN BLUE REACTION FOR FERRIC IRON

1. Take a test and control section to water
2. Treat sections with the freshly prepared acid ferrocyanide solution for 10-30 minutes
3. Wash well in distilled water.
4. Lightly stain the nuclei with 0.5% aqueous neutral red or 0.1% nuclear fast red.
5. Wash rapidly in distilled water.
6. Dehyderate, clear and mount in synthetic resin

OBSERVATIONS AND RESULTS

The present study was conducted on 364 cases of liver biopsies in the Department of pathology over a period of 5 years from 1st July 2015 to 30th June 2020 (3.5 years retrospectively and 1.5 years prospective). The age, gender, radiology and findings on clinical examination were recorded. Liver function tests that included serum alanine & aspartate aminotransferases/transaminases (AST/ALT), total bilirubin, alkaline phosphatase, total proteins & albumin levels, viral markers and other biochemical tests were analysed. Liver biopsies were studied under microscope for histomorphological features and final diagnoses were made according to the aetiology.

Out of the 364 cases there were 11 cases where no specific pathology was found on histology. Out of these in 5 cases the clinical suspicion was of autoimmune hepatitis for which autoimmune profile was negative. In 3 cases biopsies were taken intraoperatively during cholecystectomies where the surgeon visualized vague nodularity of the liver. In 3 cases radiology revealed hepatic space occupying lesions with suspicion of malignancy and liver biopsy was likely non representative.

Remaining 353 cases were analysed and divided on the basis of aetiology as shown in the table 1.

Table 1: Etiological distribution of cases (n=353).

A. Metabolic (103)	
1) Non-alcoholic fatty liver disease	**97**
2) Wilson's disease	**01**
3) Gilbert syndrome	**02**
4) Dubin Johnson syndrome	**02**
5) BRIC/PFIC	**01**
B. Drug and toxin-induced hepatotoxicity (35)	**22**
1) Alcoholic liver disease	**13**
2) Therapeutic agents (including complementary medicines and drugs of abuse)	
C. Immune mediated (71)	
1) Autoimmune hepatitis	**52**
2) Primary biliary cirrhosis	**08**
3) Primary sclerosing cholangitis	**07**
4) Overlap syndrome	**03**
5) Transplant rejection	**01**
D. Infectious (82)	
1) Viral hepatitis	
• HAV	**07**
• HBV	**50**
• HCV	**14**
• HEV	**03**
2) Tuberculosis	**08**
E. Mechanical (15)	
1) Obstructive cholestasis	**14**
2) Vascular disorders	**01**
F. Infiltrative Disorders (18)	
1) Amyloidosis	**08**
2) Sarcoidosis	**10**
G. Others (29)	
1) Neonatal giant cell hepatitis	**01**
2) Chronic hepatitis- idiopathic	**06**
3) Cholestatic liver disease- idiopathic	**05**
4) Cirrhosis – idiopathic	**17**

A. METABOLIC DISEASES

1) Non-alcoholic fatty liver disease (NAFLD)

NAFLD includes disease spectrum of Steatosis, NASH & Cirrhosis.

Age and gender distribution

Of the total 353 cases in our study, 97 cases (27.4%) were of NAFLD. The ages ranged from 11 to 80 years. Mean age of was 55 years. The most affected patients (30 cases, 30.9%) were in 51-60 years age group and no case was seen below 10 years of age.

Of the total cases, 56 of the cases were females and 41 of the cases were males with Male: Female ratio of 1:1.3

Table 2: Distribution of cases of NAFLD according to age.

Age (Years)	Number of Cases (n=97)	Percentage (%)
0-10	0	0
11-20	04	4.1
21-30	05	5.1
31-40	14	14.4
41-50	26	26.8
51-60	30	30.9
61-70	15	15.4
71-80	03	3.3

Clinical features

20 patients had history of type 2 diabetes mellitus and 25 patients were obese. Thirty-nine cases had dyslipidaemia with raised cholesterol

levels. Hepatomegaly was seen in 30 cases. Sixteen cases presented with jaundice. There was no history of any drug or alcohol intake.

Biochemical investigations

Liver function test's (LFT)

AST levels: Majority of the cases had normal levels of AST (68 cases, 70%) while raised AST levels were noted in 29 cases (30%). Eighteen cases had AST levels <2 times upper limit of normal (ULN) and 2 cases had >5 times ULN. Thus, majority of the patients (62%) had mild increase in AST levels.

ALT levels: Majority cases had normal levels of ALT (72 cases, 74.2%) and 25 cases (25.8%) had raised ALT. Nine cases (9.2%) had ALT levels <2 times ULN and 2 cases (2%) had >5 times ULN. Majority of the patients (56%) had moderate increase in ALT levels.

Total Bilirubin: Of the total 97 cases, 77 cases (79.3%) had normal total bilirubin levels and 20 cases (20.7%) had raised bilirubin levels, highest value being 23.5 mg/dl.

ALP levels: Twenty-seven cases (28%) had raised ALP levels.

Table 3: Distribution of cases of NAFLD according to Liver function tests.

LFT	Normal (n=97)		Raised (n=97)	
	No.	%	No.	%
AST (20-40 IU/L)	68	70.0	29	30.0
ALT (20-41 IU/L)	72	74.2	25	25.8
Total bilirubin (0-1.2 mg/dL)	77	79.3	20	20.7
ALP (40-129 IU/L)	70	72.0	27	28.0

Total proteins & albumin levels: Total proteins were reduced in 47 cases (48.4%) which was due to low serum albumin. In 12 cases serum albumin was low with high globulin and normal total proteins.

PTI/INR: was prolonged in 42 cases (43%).

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Autoimmune serological profile was negative in these patients.

Morphological characteristics

Classical histological changes were seen as:

- Fatty change (Steatosis)
- Ballooning degeneration
- Lobular inflammation both acute and chronic

Other changes:

- Mallory denk bodies
- Portal inflammation
- Cholestasis

These changes were analysed and graded as per CRN guidelines.

- Steatosis: All the patients (100%) had some degree of fatty change either micro vesicular or macro vesicular. Majority patients had grade 2 steatosis (42 cases, 43.2%) followed by grade 3 (29 cases 29.8%) (Picture: 1).
- Ballooning degeneration: Most of the patients had no ballooning degeneration (47 cases, 48.5%) and grade 2 ballooning degeneration was noted in 15 cases (15.5%).
- Lobular inflammation- Majority cases had grade 2 (48 cases, 49.5%) and 16 cases had grade 3 (13.5%) lobular inflammation (Picture: 2).

Masson's trichrome stain highlighted pericellular and perivenular fibrosis in majority of cases (84 cases, 86.5%). Reticulin stain highlighted maintained reticulin framework in all the cases. Perl's stain highlighted focal positivity of iron in Kupffer cells in 2 cases only. Orcein and Rhodanine stain for copper highlighted focal positivity in 2 cases.

Table 4: Distribution of cases of NAFLD according to grade of steatosis, ballooning degeneration, lobular inflammation.

Histological characteristics	Grade	Number of cases (n=97)	Percentage (%)
Steatosis	Grade 0 (<5%)	0	0
	Grade 1 (5-33%)	26	27
	Grade 2 (34-66%)	42	43.2
	Grade 3 (>66%)	29	29.8
Ballooning degeneration	Grade 0 (none)	47	48.5
	Grade 1(mild)	35	36
	Grade 2 (moderate to marked)	15	15.5
Lobular inflammation	Grade 0 (none)	00	00
	Grade 1 (<2 foci/x20 field)	33	34
	Grade 2 (2-4 foci/x20 field)	48	49.5
	Grade 3 (>4 foci/x20 field)	16	16.5

Mallory denk bodies were seen in 13 cases (13.4%) and cholestasis was seen in 20 cases (20.6%). Mild chronic lymphocytic inflammatory infiltrate was seen in the portal tracts in all the cases.

Fibrosis was analysed on Haematoxylin and eosin stain and Masson's trichrome stain and staging was done from stage 0 to stage 4.

Table 5: Distribution of cases of NAFLD according to stage of fibrosis (n=97) (Picture: 3, 4).

Stage	Number of cases	Percentage (%)
Stage 0 (no fibrosis)	13	13.4
Stage 1 (mild to moderate zone 3 perisinusoidal fibrosis)	15	15.5
Stage 2 (perisinusoidal and periportal fibrosis)	19	19.6
Stage 3(bridging fibrosis)	20	20.6
Stage 4(cirrhosis)	30	30.9

In our study, majority patients had stage 4 fibrosis (30 cases, 30.9%) and 13 cases (13.4%) had no fibrosis.

Table 6: Distribution of cases according to the spectrum of NAFLD.

Disease	n=97		Mean age (years)	M:F ratio
	No.	%		
Fatty liver disease	29	29.9	36	1:1.5
NASH	38	39.2	50	1:2
Cirrhosis	30	30.9	56	1:1.5

Analysing the spectrum of NAFLD as shown in table 6, fatty liver disease and cirrhosis constituted 29.9% and 30.9% respectively while 39.2% cases had NASH. As the disease progressed to cirrhosis the mean age of presentation also increased and there was female preponderance in all the 3 groups.

Table 7: Distribution of NAFLD cases in relation to biochemical profile.

Number of cases according to histological spectrum	Liver function test							
	Bilirubin		AST		ALT		ALP	
	Normal	Raised	Normal	Raised	Normal	Raised	Normal	Raised
Fatty liver disease (n=29)	27(93%)	02(7%)	25(86.2%)	04(12.8%)	26(89.6%)	03(10.4%)	24(82.7%)	05(17.3%)
NASH (n=38)	25(65.7%)	13(34.3%)	20(52.6%)	18(47.4%)	21(55.2%)	17(44.7%)	22(57.8%)	16(42.2%)
Cirrhosis (n=30)	20(66.6%)	10(33.4%)	24(80%)	06(20%)	25(83.4%)	05(16.6%)	24(80%)	06(20%)

Serum Bilirubin, AST, ALT and ALP were normal in majority of cases of fatty liver disease. In NASH and cirrhosis bilirubin was raised in 65.7% and 66.6% respectively. In NASH 52.6% cases had normal AST and 47.4% cases had raised AST whereas in cirrhosis 80% cases had normal AST levels and only 20% cases had raised AST. Similarly, in NASH, ALT levels were normal in 55.2% cases and raised in 44.8% cases whereas in cirrhosis 83.4% cases had normal AST and only 16.6% cases had raised levels. ALP levels were normal in 57.8% cases and raised in 42.2% cases whereas in cirrhosis 80% cases had normal ALP and only 20% cases had raised ALP.

2) Disorders of copper metabolism

Wilson's disease: There was a single case of a 28 years male who presented with jaundice.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised (174 IU/L)

ALT levels: Raised (75 IU/L)

Total Bilirubin: Raised (3.6 ug/dL) and was predominantly conjugated (2.9 ug/dL)

ALP levels: within normal limits (114 IU/L)

Total proteins & albumin levels: were within normal limits

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive.

Urinary copper (24 hrs): was raised (898.6 ug/24 hrs)

Ceruloplasmin: was raised (24.7 mg/dl)

Histological features: Liver biopsy showed distorted portal and lobular architecture with attempted nodule formation separated by thick fibrous septae. Portal tracts showed moderate infiltration by mixed inflammatory infiltrates. Interface hepatitis seen. Focal ductular proliferation was noted. Bilirubinostasis both canalicular and intracellular type with occasional bile plugs seen. Mild ballooning degeneration also noted.

Special stains: Masson's trichrome stain highlighted thin Porto- portal septae.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron was negative.

Rhodanine stain highlighted accumulation of copper pigment both in peri septal area and in centrilobular hepatocytes (Picture: 5).

3) Hereditary defects of bilirubin metabolism

3.1 Gilbert's syndrome

Age and gender prevalence

There were 2 males aged 26 years and 27 years .

Clinical features

Both the patients had on and off jaundice.

Biochemical investigations

Liver function test's (LFT)

AST, ALT, ALP levels were normal in both the cases. Total proteins & albumin levels were also normal.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive.

Histological features

Liver biopsy showed maintained portal and lobular architecture. Portal tracts were unremarkable. Hepatocytes showed presence of intracellular fine brown pigment in the hepatocytes- Lipofuscin.

Special stains

Masson's trichrome stain did not highlight fibrosis.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron was negative.

3.2 Dubin Johnson syndrome

Age and gender prevalence

There were 2 cases of the age 14 years and 22 years and both were males.

Clinical features

Both the patients presented with complain of jaundice.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised (115 IU/L and 150 IU/L)

ALT levels: Raised (126 IU/L and 158 IU/L)

Total Bilirubin: Raised (5.6 ug/dL and 5.8 ug/dL) and was predominantly conjugated (5.2 ug/dL and 5.2 ug/dL)

ALP levels: raised in both cases (220 IU/L and 480 IU/L)

Total proteins & albumin levels: within normal limits in both the cases.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

Both the liver biopsies showed maintained portal and lobular architecture. Majority of hepatocytes showed presence of abundant

coarse dark brown cytoplasmic pigment particularly in perivenular regions. Portal tracts showed mild infiltration by chronic inflammatory cells (Picture: 6).

Special stains

Masson's trichrome stain did not highlight fibrosis.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron was negative.

Orcein and Rhodanine stain for copper and copper associated protein were negative.

4) Hereditary defects of bile acid metabolism

Benign Recurrent Intrahepatic Cholestasis/Progressive Familial Intrahepatic Cholestasis

There was a single case of a 30 years male who presented with jaundice and pruritis.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised (80 IU/L)

ALT levels: Raised (87IU/L)

Total Bilirubin: Raised (8.2 ug/dL) and was predominantly conjugated (8 ug/dL)

ALP levels: within normal limits (114 IU/L)

Total proteins & albumin levels: within normal limits

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive.

Histological features

Portal tracts showed expansion with increased fibrosis and mild infiltration by mixed inflammatory infiltrates. Hepatocellular and canalicular cholestasis was seen. Focal giant cell transformation also noted. Mild ballooning degeneration and macrovesicular steatosis (<5%) was identified.

Special stains

Masson's trichrome stain highlighted thin Porto- portal septae.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron was negative.

B. DRUG AND TOXIN-INDUCED HEPATOTOXICITY

1) Alcoholic Liver Disease

Age and gender prevalence

Of the total 353 cases in our study, 22 cases (6.2%) were of ALD. The ages ranged from 21 to 70 years. Mean age was 55 years. The most affected patients (7 cases, 30.9%) were in 51-60 years age group and no case was seen below 20 years of age.

Of the total 22 cases, all were males. History of alcohol intake was present in all the cases.

Table 8: Distribution of cases according to age.

Age (Years)	Number of cases (n=22)	Percentage (%)
21-30	02	9.1
31-40	05	22.7
41-50	06	27.2
51-60	07	31.8
61-70	02	9.1

Clinical features

Only 4 patients (18%) had history of type 2 diabetes mellitus and 8 patients (36.3%) were obese. 13 patients had dyslipidaemia with raised cholesterol levels. Hepatomegaly was seen in 8 cases. Seventeen cases presented with jaundice.

Biochemical investigations

Liver function test's (LFT)

AST levels: were raised in majority (90.9%) with most (54.5%) having moderate increase (AST levels 2 times ULN to 5 times ULN). Only 1 case had AST levels >5 times ULN.

ALT levels: were raised in majority (72.7%) cases with most having

mild increase (ALT levels <2 times ULN). Only 1 case had ALT levels >5 times ULN. Reversal of AST/ALT ratio was seen in 15 cases.

Total Bilirubin: majority cases had raised bilirubin levels (13 cases, 59%) varying from 2.5mg/dL to 40mg/dL.

ALP levels: Eighteen cases (81.8%) had raised ALP levels.

Table 9: Distribution of cases according to Liver function tests (n=22).

LFT	Normal	Raised	Percentage (%)
AST (20-40 IU/L)	02	20	90.9
ALT (20-41 IU/L)	06	16	72.7
Total bilirubin (0-1.2 mg/dL)	09	13	59
ALP (40-129 IU/L)	04	18	81.8

Total proteins & albumin levels: Total proteins were reduced in 17 cases which was due to low serum albumin. In 1 case serum albumin was low with high globulin and normal total proteins.

PTI/INR: was prolonged in all cases.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Autoimmune serological profile in these patients was negative.

Morphological characteristics

Classical histological changes were seen as (Picture 7, 8):

- Fatty change (Steatosis)
- Ballooning degeneration
- Lobular inflammation both acute and chronic

Other changes:

- Mallory denk bodies
- Portal inflammation
- Cholestasis
- Perivenular fibrosis

- Neutrophilic satellitosis

These changes were analysed and graded as per CRN guidelines.

- Steatosis: All the patients (100%) had some degree of fatty change either micro vesicular or macro vesicular. Majority patients had grade 1 steatosis (12 cases, 54.5%).
- Ballooning degeneration: Most of the patients had grade 1 ballooning degeneration (10 cases, 45.4%).
- Lobular inflammation- Majority cases had grade 3 (12 cases, 54.5%) and only 3 cases had grade 2 (13.6%) lobular inflammation.

Table 10: Distribution of cases according to grade of steatosis, ballooning degeneration, lobular inflammation.

Histological characteristics	Grade	Number of cases	Percentage (%)
Steatosis	Grade 0 (<5%)	00	00
	Grade 1 (5-33%)	12	54.5
	Grade 2 (34-66%)	08	36.4
	Grade 3 (>66%)	02	09.1
Ballooning degeneration	Grade 0 (none)	07	31.8
	Grade 1(mild)	10	45.5
	Grade 2 (moderate to marked)	05	22.7
Lobular inflammation	Grade 0 (none)	00	00
	Grade 1 (<2 foci/x20 field)	07	31.9
	Grade 2 (2-4 foci/x20 field)	03	13.6
	Grade 3 (>4 foci/x20 field)	12	54.5

Mild chronic lymphocytic inflammatory infiltrate was seen in the portal tracts in all the cases.

Mallory denk bodies were seen in 12 cases (54.5%).

Cholestasis was seen in 8 cases mainly perivenular region (36.3%).

Perivenular fibrosis was seen in 10 cases (45.4%).

Fibrosis was analysed on Haematoxylin and eosin stain and Masson's trichrome stain and staging was done from stage 0 to stage 4.

Masson's trichrome stain highlighted pericellular and perivenular fibrosis in 10 cases. Reticulin stain highlighted maintained reticulin framework in 20 cases and 2 cases had collapsed reticulin framework.

Perl's stain for iron was negative in all the cases.

Orcein and Rhodanine stain for copper were not done in any of the case.

Table 11: Distribution of cases according to stage of fibrosis (n=22).

Stage	Number of cases	Percentage (%)
Stage 0(no fibrosis)	00	00
Stage 1(mild to moderate zone 3 perisinusoidal fibrosis)	01	4.5
Stage 2(perisinusoidal and periportal fibrosis)	03	13.6
Stage 3(bridging fibrosis)	05	22.7
Stage 4(cirrhosis)	13	59.2

In our study, majority patients had stage 4 fibrosis (13 cases, 59.2%) and only 1 case (4.5%) had stage 1 fibrosis.

Table 12: Distribution of cases according to spectrum of ALD (n=22).

Disease	n=22		Mean age (years)
	No.	%	
Alcoholic steatohepatitis	9	40.9	31.8
Cirrhosis	13	59.1	58.0

Analysing the spectrum of ALD as shown in table 12, majority patients (59.1%) had cirrhosis and remaining 40.9% cases had alcoholic steatohepatitis. As the disease progressed to cirrhosis mean age of presentation also increased.

Table 13: Distribution of ALD cases in relation to biochemical profile (n=22).

Number of cases according to histological spectrum	Liver function test							
	Bilirubin		AST		ALT		ALP	
	Normal	Raised	Normal	Raised	Normal	Raised	Normal	Raised
ASH (n=9)	5(55.6%)	4(44.4%)	1(11.1%)	8(88.9%)	2(22.2%)	7(77.7%)	1(11.1%)	8(88.9%)
Cirrhosis(n=13)	4(30.7%)	9(69.3%)	1(7.7%)	12(92.3%)	4(30.8%)	9(69.2%)	3(23%)	10(77%)

AST, ALT and ALP were raised in majority of cases in ASH and cirrhosis. Bilirubin was normal in 55.6% cases and raised in 44.4% patients. In cirrhosis bilirubin was raised in majority of cases and normal in 30.7% cases only.

2) Drug induced hepatotoxicity

Of the total 353 cases, 13 cases had drug induced liver injury.

Age and gender prevalence

The ages ranged from 11 to 80 years. Mean age of was 46 years. The most affected patients (4 cases, 30.7%) were in 51-60 years age group and no case was seen below 10 years of age.

Of the total 13 cases, there were 4 females and 9 males with male to female ratio of 2.5:1.

Table 14: Distribution of cases of drug induced hepatotoxicity according to age.

Age (Years)	Number of cases (n=13)
11-20	1
21-30	3
31-40	2
51-60	4
61-70	2
71-80	1

Clinical features

All patients presented with jaundice. 5 cases had history of intake of herbal medicines. 1 patient had history of levofloxacin intake, 1 patient had history of intake of amoxicillin and clavulanic acid and 1 patient had history of intake of linezolid and imipenem. 3 patients were on ATT. 2 patients also had history of some desi medication constituents of which were unknown. Based on the clinical findings, biochemical findings and histomorphological features and ruling out all other causes of liver injury diagnosis of DILI was made.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 7 cases (53.8%) highest value reaching up to 1207 IU/L

ALT levels: Raised ALT levels were noted in 7 cases (53.8%) highest value reaching up to 915 IU/L

Total Bilirubin: Raised in all the cases with highest value reaching up to 30.6 mg/dL.

ALP levels: All cases had raised ALP levels reaching up to 464IU/L.

Table 15: Distribution of cases of drug induced hepatotoxicity according to Liver function tests.

LFT	Normal (n=13)	Raised (n=13)
AST (20-40 IU/L)	3	10
ALT (20-41 IU/L)	3	10
Total bilirubin (0-1.2 mg/dL)	0	13
ALP (40-129 IU/L)	0	13

Total proteins & albumin levels: total proteins were reduced in 2 cases due to low albumin levels. In 2 cases, total albumin was low 2 cases with high globulin and normal total proteins.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

- Necro inflammatory pattern of injury was seen in 9 cases. Moderate to dense mixed inflammatory infiltrates along with eosinophils was seen in portal tracts. Mixed lobular inflammatory infiltrate was noted. Spotty and confluent necrosis was also identified.

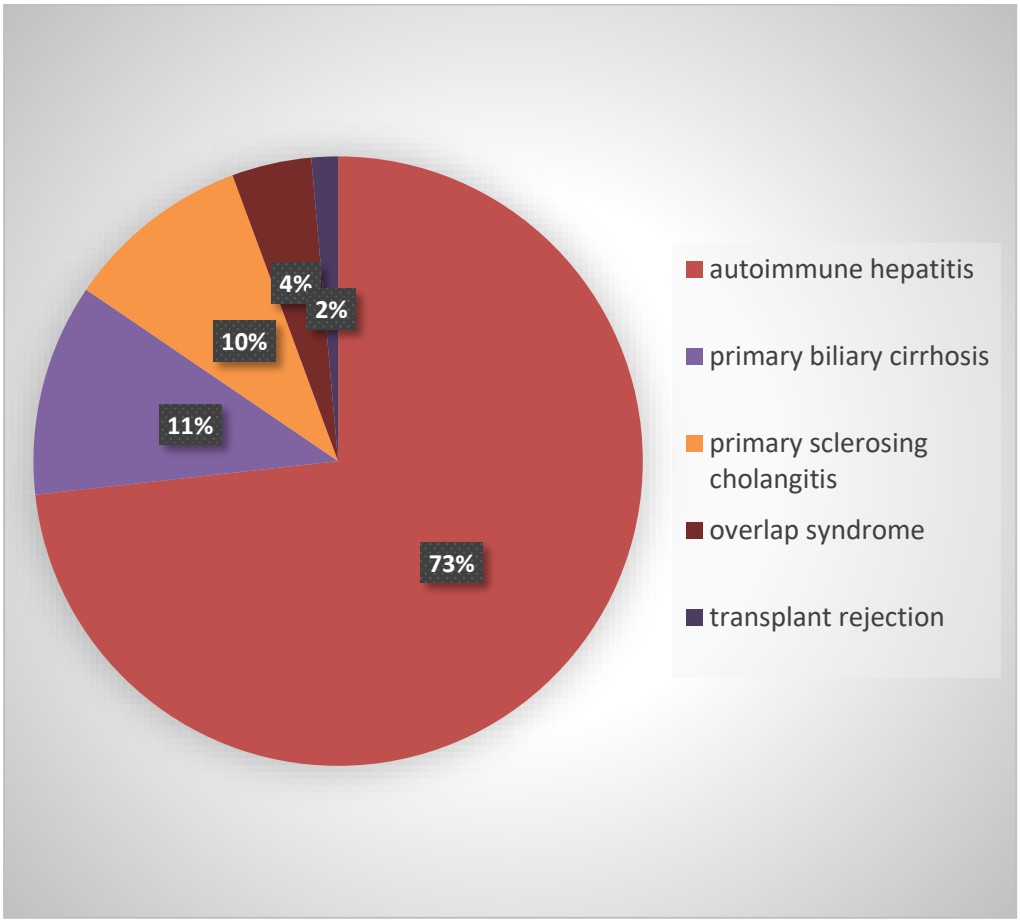
- Cholestatic pattern of injury – seen in all cases
- Steatosis- seen in 2 cases
- Fibrosis – 7 cases had increased fibrosis and 1 case had cirrhosis.

C. IMMUNE MEDIATED CAUSES OF LIVER INJURY

This category included autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, overlap syndrome and transplant rejection.

Of the total 353 cases, 71 cases had immune mediated causes of liver injury. 52 cases out of 71 cases were of autoimmune hepatitis, 8 cases were of primary biliary cirrhosis, 7 cases were of primary sclerosing cholangitis, 3 cases were of PBC- AIH overlap syndrome and 1 case of transplant rejection.

Figure 1: Distribution of cases according to causes of immune mediated liver injury.



1) **Autoimmune hepatitis**- 52 cases out of 71 cases were of autoimmune hepatitis.

Age and gender

The ages ranged from 11 to 80 years. Mean age was 55 years. The most affected patients (15 cases, 28.8%) were in 51-60 years age group and no case was seen below 10 years of age. Of the total 52 cases, 7 of the cases were males and 45 of the cases were females with Male: Female ratio of 1:6

Table 16: Distribution of cases of drug induced hepatotoxicity according to age.

Age	Cases (n-52)
11-20	05
21-30	08
31-40	09
41-50	08
51-60	15
61-70	06
71-80	01

Clinical features

Fourteen patients (26.9%) had history of type 2 diabetes mellitus. Hepatomegaly was seen in 8 cases (15.3%). Nineteen cases (36.5%) presented with jaundice.

Biochemical investigations

Liver function test's (LFT)

AST levels: Majority of the patients (37 cases, 71.1%) had raised AST levels and maximum value was 190 IU/L. Fifteen cases had normal levels of AST (28.9%).

ALT levels: Majority of the patients (31 cases, 59.6%) had raised ALT and maximum value was 175 IU/L. Only 21 cases had normal levels of ALT (40.4%).

Total Bilirubin: Majority patients had raised bilirubin levels (45 cases, 86.5%) and highest value being 38.2 mg/dl. Only 7 cases (13.5%) had normal total bilirubin levels and

ALP levels: Majority patients (32 cases, 61.5%) had raised ALP levels.

Table 17: Distribution of cases of drug induced hepatotoxicity according to Liver function tests (n=52).

LFTs	Normal		Raised	
	No.	%	No.	%
AST (20-40 IU/L)	15	29	37	71.1
ALT (20-41 IU/L)	21	40.4	31	59.6
Total bilirubin (0-1.2 mg/dL)	07	13.5	45	86.5
ALP (40-129 IU/L)	20	38.5	32	61.5

Total proteins & albumin levels: Total proteins were reduced in 4 cases (7.7%) due to low albumin. In 11 cases albumin was reduced with raised globulin and normal total proteins.

PTI/INR: was prolonged in 25 cases (48%).

Viral markers: all cases had HBsAg, anti HCV antibodies, anti-HIV antibodies negative.

Autoimmune markers: 42 cases (80.8%) of the total 52 cases had ANA positive, 33 cases (63.4%) had ASMA positive and 6 cases (11.5%) had LKM positive.

Morphological characteristics (Picture 9, 10)-

- Lymphoplasmacytic portal inflammatory infiltrates- all the cases had portal inflammation comprising of lymphocytes and plasma cells.
- Interface hepatitis – seen in 41 cases (78.8%).
- Hepatocytic rosetting was noted in 6 cases (11.5%).
- Emperipolesis was also noted in 6 cases (11.5%).

Table 18: Distribution of cases of drug induced hepatotoxicity according to stage of fibrosis.

Staging	n=52	
	No.	%
None (0)	05	9.6
Enlarged / fibrotic portal tract (1)	03	5.8
Periportal /portal-portal fibrosis (2)	05	9.6
Fibrosis with architectural distortion but no obvious cirrhosis (3)	14	26.9
Probable / definite cirrhosis (4)	25	48

Majority patients (48.1%) had definite cirrhosis and only 5.8% patients had stage 1 fibrosis. 9.6% cases had no fibrosis.

2) Primary Biliary Cirrhosis

8 cases out of 71 cases were of PBC.

Age and gender

Mean age was 37 years.

Of the total 8 cases, 7 of them were females and 1 was male with Male: Female ratio of 1:7

Biochemical investigations

Liver function test's (LFT)

AST levels: 4 cases had normal levels of AST (50%). Raised AST levels were noted in 4 cases (50%) maximum value being 174 IU/L.

ALT levels: Majority of them (6 cases, 75%) had raised ALT and maximum value was 116 IU/L. Only 2 cases had normal levels of ALT (25%)

Total Bilirubin: Majority of them (6 cases,75%) had raised bilirubin levels, highest value was 38.2 mg/dl. 2 cases (25%) had normal total bilirubin levels

ALP levels: Majority (6 cases, 75%) had raised ALP levels highest value being 549IU/mL.

Total proteins & albumin levels: Total proteins were reduced in 2 cases (25%). Hypoalbuminemia was noted in 3 cases (37.5%).

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Autoimmune markers: AMA positivity was seen in all cases.

Morphological characteristics

Of the total 8 cases, 4 cases had cholestasis. There was 1 case of PBC with granuloma formation (Picture: 11).

Table 19: Distribution of cases according to histological features of primary biliary cirrhosis.

Histological features	Number of cases (n=8)
The florid duct lesion; portal hepatitis	1
Ductular reaction and periportal hepatitis	1
Scarring; bridging necrosis, septal fibrosis	3
Cirrhosis	3

3) Primary Sclerosing Cholangitis

7 cases out of 71 cases were of PSC.

Age and gender wise prevalence

Mean age of the patients was 42 years.

Of the total 7 cases, there were 2 females and 5 males with Male: Female ratio of 2.5:1.

Biochemical investigations

Liver function test's (LFT)

AST levels: 2 cases had normal levels of AST (28.5%). Raised AST levels were noted in 5 cases (71.4%) maximum value being 145 IU/L.

ALT levels: Majority of them had normal levels of ALT (4 cases, 57.1%) and 3 cases (42.8%) had raised ALT maximum value being 132 IU/L.

Total Bilirubin: Majority (5 cases, 71.4%) had raised bilirubin levels, highest value being 25.5 mg/dl.

ALP levels: Majority of them (6 cases 85.7%) had raised ALP levels highest value being 1103 IU/mL.

Total proteins & albumin levels: Total proteins were reduced in 3 cases (42.8%) due to reduced albumin.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Autoimmune markers: ANCA positivity was seen in 4 cases (57.1%).

Morphological characteristics

Of all the 7 cases, 3 cases had periductal fibrosis

Table 20: Distribution of cases according to histological features of primary sclerosing cholangitis.

Histological features	Number of cases (n=7)	Percentage %
Portal inflammation (Lymphocytic)	1	14.3
Interface hepatitis	2	28.6
Periductal fibrosis and P-P septa	3	42.8
Cirrhosis	1	14.3

Majority of the patients had (42.8%) had periductal fibrosis and porto-portal septae. Interface hepatitis was seen in 28.6% cases only.

4) PBC-AIH overlap syndrome

There were 3 cases of overlap syndrome with mean age of 37 years. All the patients were females and clinical presentation was jaundice.

AST levels: 2 cases had raised AST levels maximum value being 174 IU/L.

ALT levels: 2 cases had raised ALT maximum value being 116 IU/L.

Total Bilirubin: All the cases had raised bilirubin levels, highest value being 38.8 mg/dl.

ALP levels: 1 case had raised ALP levels.

Total proteins & albumin levels: Normal in all cases.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Autoimmune markers: AMA, ANA and ASMA was positive in all the cases.

Morphological characteristics

There was seen portal and lobular inflammation by chronic inflammatory infiltrates, interface hepatitis and ductular reaction.

5) Transplant rejection

There was a single case of acute cellular rejection of a 48 years old male.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels maximum value being 506 IU/L.

ALT levels: Raised ALT maximum value being 1492 IU/L.

Total and direct bilirubin: Raised in both the cases (8.8 mg/dL and 7.3 mg/dL).

ALP levels: raised ALP levels.

Total proteins & albumin levels: Reduced.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive.

Morphological characteristics

Liver biopsy showed portal tract expansion and infiltration by mixed inflammatory infiltrates comprising of lymphocytes, neutrophils and eosinophils with focal spill over into periportal parenchyma. Portal vein tributaries in most of tracts showed subendothelial infiltration by lymphocytes with lifting and focal disruption of endothelium. Few of the bile ducts were cuffed and infiltrated by lymphocytic infiltrate. In the

centrilobular area (Zone-3) there was prominent perivenular hepatocyte necrosis. Acidophilic bodies noted along with focal sinusoidal dilatation.

Table 21: Banff grading- Rejection activity index (RAI).

Category	Score
Portal inflammation	3
Bile duct inflammation/ damage	1
Venular endothelial inflammation	3

Global assessment rejection Grade III (Severe)

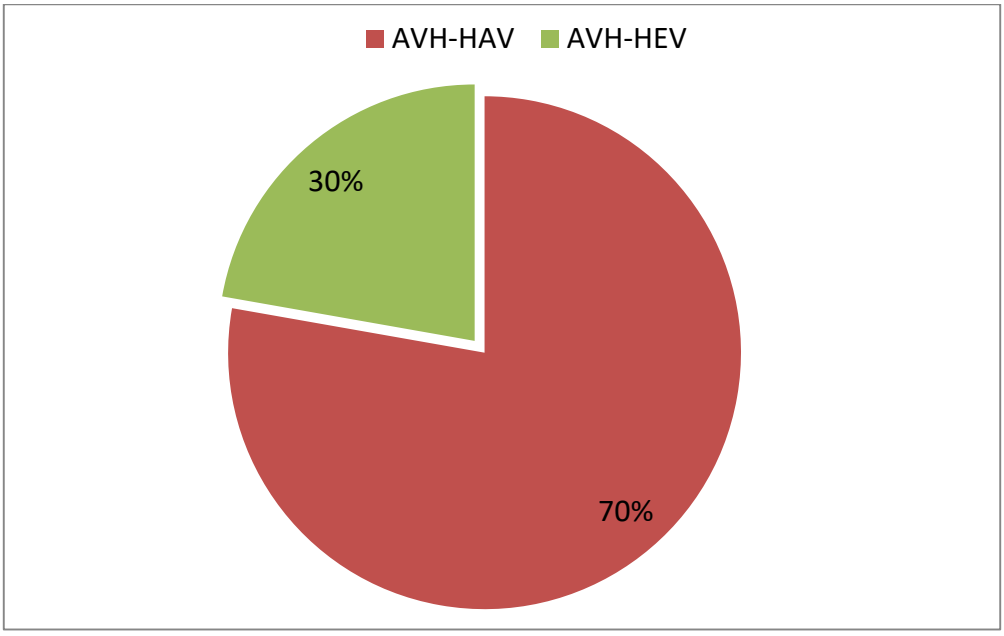
D. INFECTIOUS CAUSES OF LIVER INJURY

1) Viral hepatitis

Of the total 353 cases, 74 cases had viral hepatitis. Ten cases were of acute viral hepatitis and 64 cases were of chronic viral hepatitis.

1.1 Acute viral hepatitis- Seven cases were caused by hepatitis A virus and 3 cases were caused by hepatitis E virus.

Figure 2: Distribution of cases according to aetiology of acute viral hepatitis.



Age and gender wise prevalence

Mean age was 34 years.

Of the total 10 cases, 7 of the cases were males and 3 of the cases were females with Male: Female ratio of 2:1

Table 22: Distribution of cases of acute viral hepatitis according to age.

Age(years)	Number of cases(n=10)
10-20	2
21-30	2
30-40	2
41-50	2
51-60	1
61-70	1

Clinical features

All cases presented with complain of jaundice and fever. In 3 cases there was hepatic encephalopathy. There was no history of any drug or alcohol intake.

Biochemical investigations

Liver function test's (LFT)

AST levels: All cases had raised AST levels maximum value being 540 IU/L.

ALT levels: All cases had raised ALT levels maximum value being 582 IU/L.

Total Bilirubin: All cases had raised bilirubin highest value being 25 mg/dl.

ALP levels: All seven cases had raised ALP levels reaching up to 461 IU/L.

Total proteins & albumin levels: Total proteins were reduced in 3 cases (30%) due to low albumin.

PTI/INR: PTI/INR was prolonged in 3 cases (30%).

Viral markers: AntiHAV antibodies were positive in 7 cases and anti HEV antibodies were positive in 3 cases. HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Morphological characteristics

- Marked lobular disarray was seen in 5 cases.
- Ballooning degeneration- mild to moderate ballooning degeneration was seen in all cases.
- Spotty and confluent necrosis- was seen in 7 and 4 cases respectively.
- Portal inflammation – Three cases had mild to moderate infiltration by chronic inflammatory infiltrates and 2 cases had dense acute and chronic inflammation.
- Cholestasis – seen in all cases. Acidophilic bodies- seen in 3 cases.

Special stains

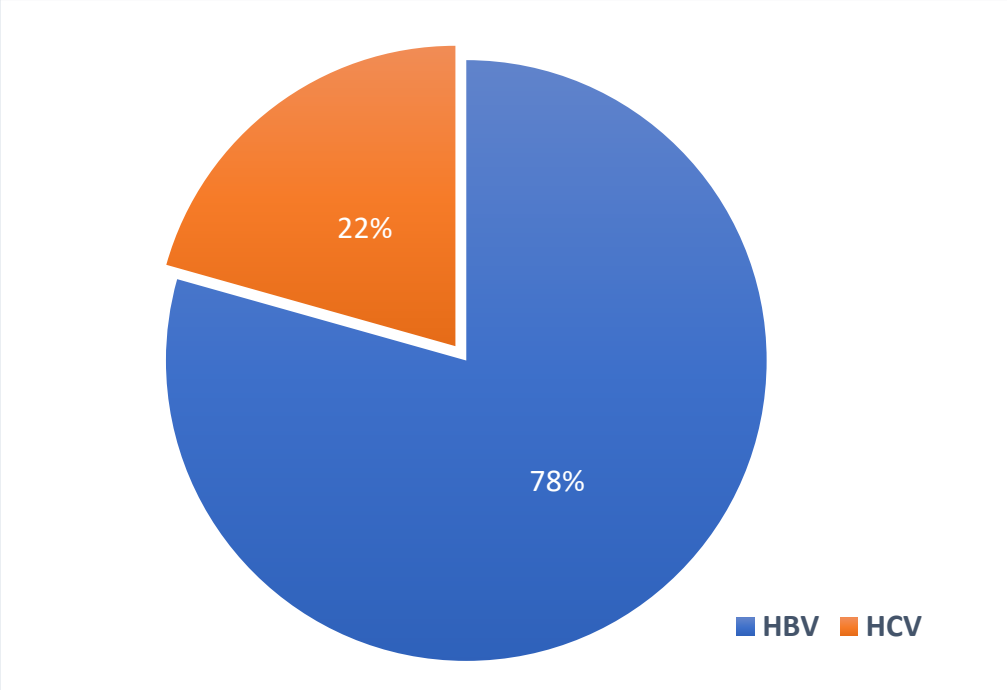
Masson's trichrome stain did not highlight fibrosis.

Reticulin stain highlighted collapse of reticulin framework in 3 cases.

Perls stain for iron was negative.

1.2 Chronic viral hepatitis – 64 cases had chronic viral hepatitis. 50 cases were caused by HBV infection and 14 cases were caused by HCV infection.

Figure 3: Distribution of cases according to aetiology of chronic viral hepatitis.



Age and gender wise

The ages ranged from 11 to 80 years. The most affected patients (21 cases, 32.8%) were in 21-30 years age group and no case was seen below 10 years of age.

Of the total 50 cases of CVH caused by HBV, 36 were males and 14 females with Male to female ratio of 2.5:1. Of the total 14 cases of CVH caused by HCV there were 10 males and 4 females with Male: Female ratio of 2.5:1.

Table 23: Distribution of cases of chronic viral hepatitis according to age.

Age (years)	Number of cases (n=64)	
	HBV (50)	HCV (14)
11-20	02	1
21-30	20	1
31-40	07	4
41-50	10	2
51-60	05	5
61-70	05	0
71-80	01	1

Clinical features

Hepatomegaly was seen in 6 cases. Eleven cases presented with jaundice. There was history of significant alcohol intake in 3 cases. Autoimmune serological profile in these patients was negative.

Biochemical investigations

Liver function test's (LFT)

AST levels: Majority of the patients had raised AST levels (48 cases 75%) maximum value being 190 IU/L. Normal levels of AST was seen in 16 cases (25%).

ALT levels: Majority cases (43 cases, 67.2%) had raised ALT maximum value being 175 IU/L. 21 had normal levels of ALT (32.8%)

Total Bilirubin: Majority cases 39 cases (60.9%) had normal total bilirubin levels and 25 cases (39%) had raised bilirubin levels, highest value being 18.2 mg/dl.

ALP levels: 23 cases (35.9%) had raised ALP levels.

Table 24: Distribution of cases of chronic viral hepatitis according to Liver function tests (n=64).

LFTs	HBV (n=50)		HCV (n=14)	
	Normal	Raised	Normal	Raised
AST (20-40 IU/L)	06	44	10	04
ALT (20-41 IU/L)	13	37	08	06
Total bilirubin (0-1.2 mg/dL)	29	21	10	04
ALP (40-129 IU/L)	32	18	09	05

Total proteins & albumin levels: Total proteins were reduced in 37 cases (57.8%) due to low albumin. Hypoalbuminemia was noted in 5 cases where total total proteins were normal and globulin levels were high.

PTI/INR: 27 cases (42.2%) had prolonged value.

Viral markers: HBsAg and HBV DNA were positive in all 50 cases. Anti HCV antibodies were positive in all 11 cases. Anti-HIV was non-reactive in all the cases.

Morphological characteristics

HBV

- Ground glass hepatocytes- 9 cases had ground glass appearance (Picture: 12)

HCV

- Steatosis – 2 cases had macro vesicular steatosis

Table 25: Distribution of cases of chronic viral hepatitis showing scoring according to Histologic activity index (HAI) Necro-Inflammatory Score.

	HBV (n=50)	HCV (n=14)
Interface hepatitis		
Absent (0)	35	6
Mild (1)	08	4
Mild/moderate (2)	05	2
Moderate (3)	02	2
Severe (4)	00	0
Confluent necrosis		
Absent	34	4
Focal confluent	05	3
Zone 3 necrosis in some	03	2
Zone 3 necrosis in most	02	2
Zone 3 necrosis + occasional portal to central bridging	04	3
Zone 3 necrosis+ multiple portal to central bridging	01	0
Pan acinar/multinecrosis	01	0
Focal lytic necrosis		
Absent	30	5
<1 focus/10x	08	5
2-4 focus/10x	06	3
5-10 focus/10x	04	1
>10 focus/10x	02	0
Portal inflammation		
None	15	0
Mild some or all portal tracts	18	8
Moderate some or all portal tracts	08	4
Moderate to marked	05	2
Marked	04	0

Most of the cases of HBV and HCV had mild interface hepatitis (8 cases of HBV and 4 cases of HCV). Focal confluent necrosis was seen in majority of cases (5 cases of HBV and 3 cases of HCV). Focal lytic necrosis was seen in 8 cases of HBV and 5 cases of HCV. Mild portal inflammation was noted in most of the cases (18 cases of HBV and 8 cases of HCV)

Table 26: Distribution of cases of chronic viral hepatitis showing grading according to Histologic activity index (HAI) Necro-Inflammatory Score.

Grade	Score	HBV (n=50)	HCV (n=14)
Minimal chronic hepatitis	1-3	37	6
Mild chronic hepatitis	4-8	12	8
Moderate chronic hepatitis	9-12	01	0
Severe	13-18	00	0

Most of the patients of HBV had minimal chronic hepatitis (37 cases) and most of the patients of HCV had mild chronic hepatitis.

Table 27: Distribution of cases of chronic viral hepatitis showing staging according to Metavir system.

Staging	HBV (n=50)	HCV (n=14)
None (0)	15	0
Enlarged / fibrotic portal tract (1)	18	6
Periportal /portal-portal fibrosis (2)	10	4
Fibrosis with architectural distortion but no obvious cirrhosis (3)	04	2
Probable / definite cirrhosis (4)	03	2

Most of the patients of HBV and HCV had stage 1 fibrosis (18 cases of HBV and 6 cases of HCV)

2) Tuberculosis

Of the total 353 cases, 8 cases had tuberculosis.

Age and gender prevalence

Mean age was 53 years.

Of the total 8 cases, there were 3 females and 5 males with male to female ratio of 1:1.6.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 3 cases (37.5%) highest value reaching up to 150 IU/L

ALT levels: Raised ALT levels were noted in 3 cases (37.5%) highest value reaching up to 160 IU/L

Total Bilirubin: Raised in 2 cases with highest value reaching up to 8.2 mg/dL.

ALP levels: 4 cases had raised ALP levels reaching up to 1167 IU/L.

Table 28: Distribution of cases of tuberculosis according to Liver function tests.

LFT	Normal (n=8)	Raised (n=8)
AST (20-40 IU/L)	5	3
ALT (20-41 IU/L)	5	3
Total bilirubin (0-1.2 mg/dL)	6	2
ALP (40-129 IU/L)	4	4

Total proteins & albumin levels:

Total proteins were reduced in 1 case (12.5%). Albumin was reduced in 3 cases (37.5%).

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

Well formed, large epithelioid cell granulomas with lymphocytic collar were seen in 5 cases out of the 8 cases diagnosed as tuberculosis. Rest of the 3 cases had caseous necrosis, langhan's type giant cells and foreign body giant cells which gave the clue of tubercular aetiology (Picture: 13).

Special stains

Masson's trichrome stain did not highlight increased fibrosis

Reticulin stain highlighted maintained reticulin framework and was not accentuated around the granulomas.

20% ZN stain and iron were negative in all cases.

E. OBSTRUCTIVE CHOLESTASIS

1) Mechanical causes

There were 14 cases where obstruction was the main pathology.

Age and gender prevalence

Mean age was 51 years.

There were 9 males and 5 females with male to female ratio of 1.8:1.

Clinical features

3 patients presented with complain of jaundice. Ultrasound was available for 2 patients and 1 had cholestatic liver disease and 1 had obstructive biliopathy.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 3 cases (21.4%) highest value reaching up to 211 IU/L

ALT levels: Raised ALT levels were noted in 3 cases (21.4%) highest value reaching up-to 190 IU/L

Total Bilirubin: Raised in all the cases highest value being 15.2 ug/dL and was predominantly conjugated.

ALP levels: raised in 4 cases (28.6%) highest value being 1507 IU/L.

Total proteins & albumin levels: Total proteins were reduced in 1 case. Albumin was reduced in 2 cases.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

Cholestasis was present seen as bile plugs seen in 8 cases, 7 cases had ductular reaction and ductular proliferation. 8 cases had cirrhosis.

Special stains

Masson's trichrome stain highlighted nodule formation in 8 cases, 2 cases had expanded portal tracts with increased fibrosis.

Reticulin stain highlights maintained reticulin framework in 13 cases and 1 case had collapsed reticulin framework.

Perl's stain for iron was focally positive in few of the Kupffer cells (Grade 2) in 1 case and negative in 13 cases (Picture: 14).

2) Budd Chiari syndrome

There was a single case of 45 years male whose ultrasound abdomen revealed veno occlusive disease. Total bilirubin was raised (3.8mg/dL). ALT, AST levels, albumin and total proteins were normal.

Histological features

Liver biopsy showed distorted portal and lobular architecture with attempted nodule formation. There was sinusoidal dilatation with perisinusoidal fibrosis. Centrilobular hepatocytes showed atrophy with focal drop out. Portal tracts had increased fibrosis with patent portal veins. Cavo-caval fibrous septa identified.

Special stains

Masson's trichrome stain highlighted nodule formation and perivenular and perisinusoidal fibrosis.

Reticulin stain highlighted reticulin rich areas.

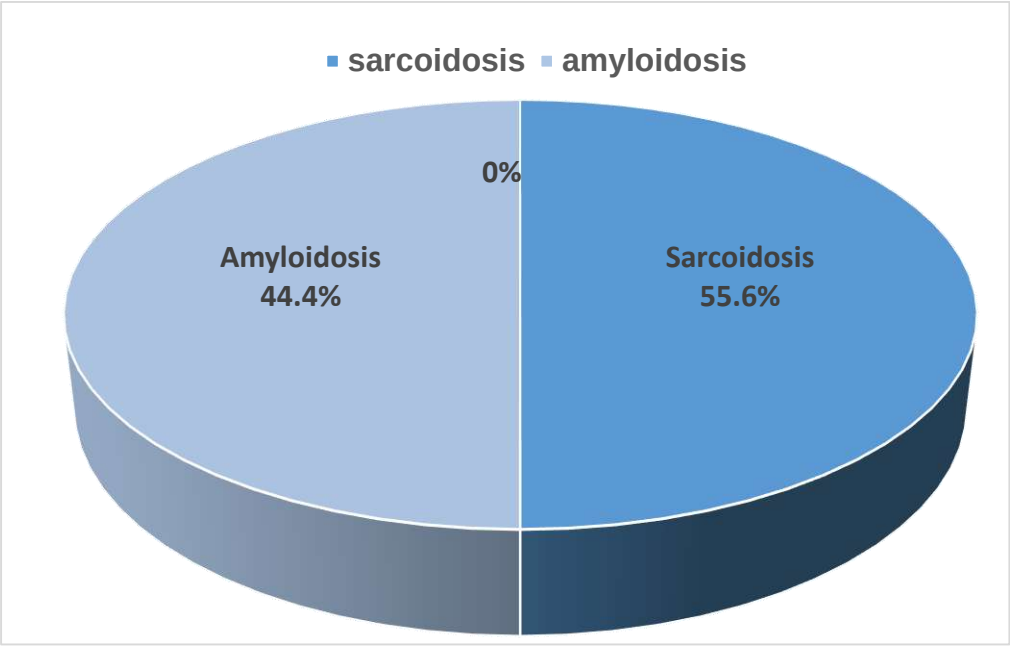
Perl's stain for iron negative.

F. INFILTRATIVE LIVER DISEASES

Sarcoidosis and amyloidosis were the infiltrative liver diseases included in my study.

There were 10 cases of sarcoidosis and 8 cases of amyloidosis.

Figure 4: Distribution of infiltrative diseases.



1) Sarcoidosis

Age and gender prevalence

Mean age of the patients was 54 years. Of the total 10 cases, there were 6 females and 4 males with male to female ratio of 1:1.2.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 4 cases (40%) highest value reaching up to 250 IU/L

ALT levels: Raised ALT levels were noted in 4 cases (40%) highest value reaching up to 275 IU/L

Total Bilirubin: Raised in 2 cases with highest value reaching up to 13.6 mg/dL.

ALP levels: All cases had raised ALP levels reaching up to 552 IU/L.

Table 29: Distribution of cases of sarcoidosis according to Liver function tests.

LFT	Normal (n=10)	Raised (n=10)
AST (20-40 IU/L)	6	04
ALT (20-41 IU/L)	6	04
Total bilirubin (0-1.2 mg/dL)	8	02
ALP (40-129 IU/L)	0	10

Total proteins & albumin levels: Total proteins were reduced in 2 cases (20%). Albumin was reduced in 2 cases (20%).

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

ACE levels: Normal range was taken as 65-115 U/L. It was raised in 5 cases (50%) highest value being 316 U/L.

Histological features

Tiny multiple epithelioid cell granulomas seen in the parenchyma. Granulomas were naked with no lymphocytic collar. Foreign body giant cell reaction seen. Granulomas were surrounded by fibrosis in 3 cases (Picture: 15A).

Special stains

Masson's trichrome stain highlighted the increased fibrosis.

Reticulin stain highlighted maintained reticulin framework and was accentuated around the granulomas in 5 cases (Picture: 15B).

20% ZN stain and perl's stain were negative in all cases.

2) Amyloidosis

Age and gender prevalence

Of the total 8 cases, mean age of the patients was 58 years. Of the total 8 cases, there were 3 females and 5 males with male to female ratio of 1:1.2.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 4 cases (50%) highest value reaching up to 75 IU/L

ALT levels: Raised ALT levels were noted in 4 cases (50%) highest value reaching up to 80 IU/L

Total Bilirubin: Raised in 2 cases with highest value reaching up to 8.5 mg/dL.

ALP levels: 6 cases had raised ALP levels reaching up to 362 IU/L.

Table 30: Distribution of cases of amyloidosis according to Liver function tests.

LFT	Normal (n=8)	Raised (n=8)
AST (20-40 IU/L)	4	4
ALT (20-41 IU/L)	4	4
Total bilirubin (0-1.2 mg/dL)	6	2
ALP (40-129 IU/L)	0	8

Total proteins & albumin levels: Total proteins were reduced in 3 cases (37.5%). Albumin was reduced in 3 cases (37.5%).

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

All the cases showed presence of deposition of pink acellular eosinophilic material seen in various patterns as sinusoidal, vascular and mixed pattern. In 1 case with mixed pattern eosinophilic acellular globular structures were seen and also in portal tracts. 1 case had cirrhosis (Picture: 16A&B).

Table 31: Distribution of cases of amyloidosis according to histological features (n=8).

Pattern	Number of cases
Sinusoidal pattern (linear)	4
Vascular pattern	1
Mixed sinusoidal & vascular pattern	3

Special stains

Congo red stain was positive in all the cases. Polarizing microscopy was done which showed apple green birefringence (Picture: 16C).

G. OTHER LESIONS

1) Neonatal giant cell hepatitis

There was 1 case of a 6-year-old female who presented with complain of jaundice and mild hepatomegaly. Total and direct bilirubin were raised (total bilirubin-38.4mg/dL). AST, ALT and ALP were raised. Total protein and bilirubin were normal.

Histological features

Liver biopsy showed marked lobular disarray. Hepatocytes showed rosetting, giant cell transformation and marked feathery degeneration. Portal tracts showed mild chronic inflammatory infiltrates. No increase in fibrosis was seen**.**

2) Chronic hepatitis- Idiopathic

There were 6 cases of chronic hepatitis aetiology of which were unknown.

Age and gender prevalence

Mean age was 53 years.

Of the total 6 cases, there were 5 females and 1 male with male to female ratio of 1:5

Clinical features

4 patients presented with complain of jaundice. Ultrasound was available for 3 patients suggestive of chronic liver disease.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 4 cases (66.7%) highest value reaching up to 146 IU/L

ALT levels: Raised ALT levels were noted in 4 cases (66.7%) highest value reaching up to 118 IU/L

Total Bilirubin: Raised in 2 cases with highest value reaching up to 26.6 mg/dL.

ALP levels: 2 cases had raised ALP levels reaching up to 210 IU/L.

Table 32: Distribution of cases of chronic hepatitis- idiopathic according to Liver function tests.

LFT	Normal (n=6)	Raised (n=6)
AST (20-40 IU/L)	2	4
ALT (20-41 IU/L)	2	4
Total bilirubin (0-1.2 mg/dL)	4	2
ALP (40-129 IU/L)	4	2

Total proteins & albumin levels: Total proteins were normal in all cases. Albumin was normal in all cases.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

Liver biopsy showed maintained portal and lobular architecture in 2 cases and was distorted in 4 cases with increased portal fibrosis and Porto portal septa in 1 case. Portal tracts showed moderate to dense infiltration by chronic inflammatory infiltrates in all the cases.

Special stains

Masson's trichrome stain highlighted increased portal fibrosis.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron was negative.

3) Cholestatic liver disease- Idiopathic

5 cases had cholestatic liver disease.

Age and gender prevalence

Mean age was 53 years.

Of the total 5 cases, there were 1 female and 4 males with male to female ratio of 1:4

Clinical features

4 patients presented with complain of jaundice. Ultrasound was available for 1 patient suggestive of cholestatic liver disease.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 3 cases (60%) highest value reaching up to 194 IU/L

ALT levels: Raised ALT levels were noted in 3 cases (60%) highest value reaching up to 97 IU/L

Total Bilirubin: Raised in 4 cases (80%) with highest value reaching up to 14.2mg/dL.

ALP levels: 2 cases had raised ALP levels reaching up to 600 IU/L.

Table 33: Distribution of cases of cholestatic liver disease according to Liver function tests.

LFT	Normal (n=5)	Raised (n=5)
AST (20-40 IU/L)	02	03
ALT (20-41 IU/L)	03	02
Total bilirubin (0-1.2 mg/dL)	01	04
ALP (40-129 IU/L)	03	02

Total proteins & albumin levels: within normal limits.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

Liver biopsy showed maintained portal and lobular architecture in all the cases. Portal tracts showed moderate infiltration by chronic inflammatory infiltrates in all the cases. Hepatocytic rosetting was seen in 2 cases and other 3 cases showed mild ballooning degeneration. 2 cases had intracellular and canalicular cholestasis along with bile plugs.

Special stains

Masson's trichrome stain highlighted Grade 1 fibrosis in 2 cases. No increase in fibrosis was seen in remaining 3 cases.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron was negative in all cases.

4) Cirrhosis – idiopathic

There were 17 cases of cirrhosis cause of which was unknown.

Age and gender prevalence

Mean age was 53 years.

Of the total 17 cases, there were 9 females and 8 males with male to female ratio of 1:1.2

Clinical features

8 patients presented with complain of jaundice. Ultrasound was available for 3 patients suggestive of chronic liver disease.

Biochemical investigations

Liver function test's (LFT)

AST levels: Raised AST levels were noted in 8 cases (47%) highest value reaching up to 146 IU/L

ALT levels: Raised ALT levels were noted in 9 cases (52.9%) highest value reaching up to 118 IU/L

Total Bilirubin: Raised in 9 cases (52.9%) with highest value reaching up to 32.4 mg/dL.

ALP levels: 3 cases had raised ALP levels reaching up to 514 IU/L.

Table 34: Distribution of cases cirrhosis according to Liver function tests.

LFT	Normal (n=17)	Raised (n=17)
AST (20-40 IU/L)	9	8
ALT (20-41 IU/L)	10	7
Total bilirubin (0-1.2 mg/dL)	9	8
ALP (40-129 IU/L)	14	3

Total proteins & albumin levels: Total proteins were reduced in 3 cases. Albumin was reduced in 4 cases.

Viral markers: HBsAg, anti HCV antibodies, anti-HIV were non-reactive in all the cases.

Histological features

Liver biopsy showed distorted portal and lobular architecture with nodule formation. Portal tracts showed moderate to dense infiltration by chronic inflammatory infiltrates in all the cases.

Special stains

Masson's trichrome stain highlighted nodule formation.

Reticulin stain highlighted maintained reticulin framework.

Perl's stain for iron highlighted hemosiderin in Kupffer cells in 1 case. 15 cases had negative iron stain.

Chronic liver disease was seen in 57% cases, acute liver disease in 4.5%, granulomatous liver disease in 5% cases and others constitute 33.5% cases.

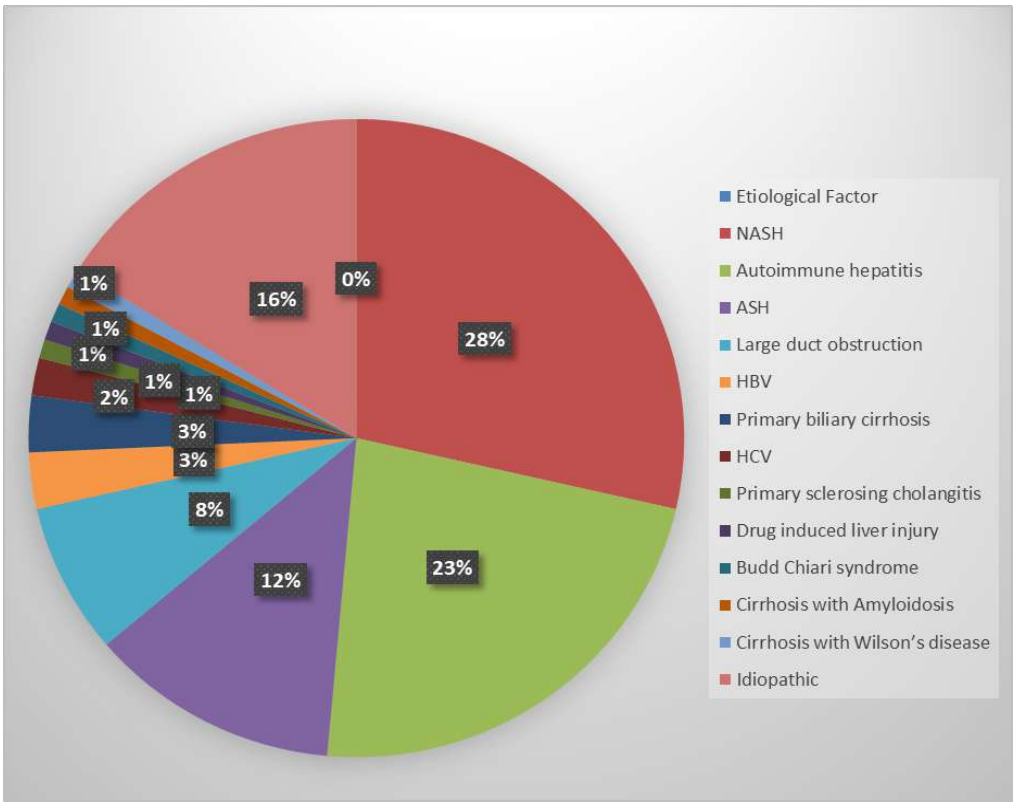
All 353 cases have been also classified according to the pattern of injury observed on histopathology as depicted in Table 35.

Cirrhosis was observed in 105 patients out of total 353 cases. We have distributed these cases according the underlying etiology as given in table 35. Majority of the cases were NASH related followed by AIH. No cause was identified in 17 cases inspite of extensive investigations.

Table 35: Distribution of cases according to causes of cirrhosis (n=105).

Etiological Factor	Number of cases	Percentage (%)
NASH	30	28.8
Autoimmune hepatitis	24	23.0
ASH	13	12.5
Large duct obstruction	8	7.6
HBV	3	2.8
Primary biliary cirrhosis	3	2.8
HCV	2	1.9
Primary sclerosing cholangitis	1	0.9
Drug induced liver injury	1	0.9
Budd Chiari syndrome	1	0.9
Cirrhosis with Amyloidosis	1	0.9
Cirrhosis with Wilson's disease	1	0.9
Idiopathic	17	16.1

Figure 5: Distribution of cases according to cause of cirrhosis.



DISCUSSION

Liver disease is a major cause of morbidity and mortality in the world and in India. Multiple etiological factors lead to a similar clinico-pathological syndrome especially in CLD.

In our study there were 353 cases having liver pathology which were then analysed and classified on the basis of aetiology. It was found that chronic liver disease was seen in 57% cases. NASH was seen in 23.7% cases followed by autoimmune hepatitis in 13.3% cases, hepatitis B in 9.9%, hepatitis C in 3.9 % and alcoholic liver disease in 6.2% cases. This was discordant with a prospective, multicentric study to find the aetiology and clinical profile of chronic liver disease in India conducted by Mukherjee et al[45]. Data was collected from Feb, 2010 to Jan, 2013 and 13014 cases of chronic liver disease were analysed. According to their study hepatitis B was the most common cause of chronic liver disease seen in 33.3% cases followed by hepatitis C seen in 21.5% cases and ALD in 17% cases. Another study was conducted by Khokhar et al[46] in a hospital of Pakistan on 518 liver biopsies. Of these 86% had hepatitis C and 10.7% had hepatitis B. Thus, chronic hepatitis C made the largest percentage in their series of cases. The reason for lower percentage of chronic viral hepatitis leading to chronic liver disease in our study is because according to most HBV and HCV treatment guidelines, liver biopsy has been reserved for cases having high viral load and persistently elevated liver enzyme. For rest of the cases fibro scan is used to monitor disease progression.

In our study NASH was the cause of cirrhosis in 28.8% cases followed by autoimmune hepatitis in 23% and ASH in 12.5% cases. Our study was comparable to a Chinese study done by Wang et al.

Table 36-Distribution of cases according to etiology of cirrhosis.

Study	ASH	HCV	HBV	NASH	Misc.
Our study	12.5%	2%	1%	28.8%	55.7%
Mukerjee et al[45]	33.8%	17.3%	18.1%	1.7%	29.1%
Sharma et al[47]	62.9%	2.8%	10.1%	7.9%	16.3%
Goyal et al[48]	49.2%	29.5%	3.9%	13.7%	3.7%
Wang et al[49]	4.5%	1%	7.1%	15%	72.4%

NAFLD- It is an important cause of liver disease burden across the world and is not just a disease of western world. By definition, although the histopathological features of NAFLD are identical to other causes of liver diseases, its diagnosis require absence of alcohol intake and other causes of chronic liver disease.

Although NAFLD has been described in all ages but usually prevalence increases with age[50,51,52].It has also been shown likelihood of progressive disease and mortality in older patients with NAFLD.

In our study, 76% cases with NAFLD were over >40 years and only 24% were younger which was similar to a study done by Frith et al that showed prevalence in higher age group[51]. In our study, there were 56 females and 41 males with Male:Female ratio of 1:1.3. This showed that there was female predilection which was similar to the study done by Guy et al[53] and Ajay et al[54]. Studies conducted by Singh et al[55] and Swiderska-syn et al[56] showed male preponderance.

Majority of the cases had normal levels of AST and ALT (70% and 74%).Total Bilirubin was normal in 77 cases (79%) and only 20 cases (21%) had raised bilirubin levels, highest value being 23.5 mg/dl. Twenty-seven cases (27.8%) had raised ALP levels. Similar findings have been described by Mandal et al[14] who has also described normal LFTs in most of the patients.

The histological spectrum of fatty liver disease includes steatosis, hepatocellular injury (ballooning degeneration, Mallory hyaline bodies), inflammation and fibrosis of varying degree. Key characteristic histological features in NAFLD are located in centrilobular location which may be consistent with metabolic zonation within liver acinus[57].

In our study majority patients had grade 2 steatosis (43.2%) followed by grade 3 steatosis (29.8%). Our findings were consistent with the study done by Takahashi et al[58] in Japan were also majority patients had grade 2 steatosis.

Hepatocyte ballooning degeneration which is considered a hallmark of steatohepatitis, was appreciated in lesser frequency in present study as compared to previous studies[57,59] specially grade 2 which may be due to geography and ethnicity of Indian patients.

Most of the subjects in our study had high grade lobular inflammation (grade 2 and 3) while studies done by Takahashi et al[58] and Rakha et al[60] showed contrasting features where grade 1 was predominant.

Majority of our patients (51.5%) had high grade fibrosis (stage 3 and 4) and 35% patients had stage 1 and 2 fibrosis. 13% patients had no fibrosis. This was discordant with the study conducted by Singh et al[61] in which sample size was only 60. In his study majority patients (70%) had stage 1 and 2 fibrosis and only 4% patients had stage 3 and 4 fibrosis. This might be due to late presentation of the patients. Also, the sample size of above stated study was less as compared to the present study.

Alcoholic liver disease-In the present study, majority of the patients (68%) were >40 years of age which is in concordance with the study done by Ray et al[62] over a total of 224 patients while in a study done by Chavan et al[63] majority of patients were a decade less. The presence of high age in our patients can be explained by the fact that most of the times alcoholics are asymptomatic and so when patient presents with deranged LFTs disease has already progressed.

All the patients in our study were males as expected which is in concordance with almost all the studies done on alcoholic liver disease. This male predominance may be due to prevalent socioeconomic

factors especially as drinking being taboo by women in Indian population.

In the present study, bilirubin was raised in 59 % (>1.2 mg/dl) cases. Serum bilirubin ranged from 0 to 1.2 mg/dl. AST levels were elevated in 90.9% patients and ALT in 68%. Almost similar pattern of elevated liver enzymes were also seen in study done by Khatroth et al[64] in a south medical college. In the present study, most common presentation was jaundice seen in 77% patients. Similar findings were reported by Pathak et al[65] where most commonly observed clinical finding was jaundice followed by hepatomegaly. By a study conducted by Chavan et al[65] jaundice was seen in 88% cases being similar to the present study.

In the present study, either macrovesicular or microvesicular steatosis or both were present in all the patients and most of the patients had grade 1 steatosis (54.5%). This was in concordance with the study conducted by Singh et al[63] in which also 55.3% patients had grade 1 steatosis. 45.5% patients had grade 1 ballooning degeneration in our study similar to the study done by Rakha et al[62]. Lobular inflammation was seen in 54.5% cases in the present study although the above stated studies had most of the patients with grade 1 inflammation. Mallory denk bodies were seen in 54.5% cases similar to the study done by Rakha et al[62] where also only 19% patients had Mallory denk bodies.

When comparing the histological profile of the patients of NASH and ASH both had steatosis. It was prominent and mainly grade 2 and 3 in 63% patients of NASH and only 45.3% of ASH. Ballooning degeneration of hepatocytes was more in ASH patients compared to NASH (ASH vs NASH ballooning grade 1 and 2 – 68.2% vs 51.4%). There was no significant difference in lobular inflammation. Mallory denk bodies were much more frequent and prominent in ASH compared to NASH (ASH vs NASH- 54.5% and 9.2%). Pericellular and perisinusoidal fibrosis was more common in NASH (NASH vs ASH - 34.9% vs 18.1%). Cirrhosis was more common ASH (ASH vs NASH-

59% vs 30%). Similar findings were seen in study done by Singh et al[63] who did a comparative study of NASH and ASH.

Drug induced liver injury remains the most common cause of acute liver failure. Excluding overdose, nearly all causes of DILI that are seen are found to be idiosyncratic in nature because affected individuals represent only small population of those being treated with such drugs. In the absence of other diagnostic tests or other biomarkers the diagnosis of DILI require high index of suspicion after excluding other causes of abnormal liver tests. In the present study, most of the patients were in older age group >50 years. This is similar to the study done is Iceland in which predominant cases of DILI were in older age group[66]. This increased incidence in older patients might be attributed to the increased prescription rate of drugs in older patients. In the present study 69% patients were males and 31% patient were females. It is discordant with several previous studies done by American DILI network[67] which showed prevalence of females in 59% of cases. However, it is in concordance with Spanish DILI registry[68] were also male predominance was seen. The main reason for the difference might be due to different drug pharmacokinetics/ dynamics or hormonal effects on the drug metabolites. However, it is consistently observed in our study that women tend to be younger while males developing DILI tend to be older as also studied by Lucena et al[69].

Autoimmune hepatitis- In the present study male to female ratio was 1:6 and the mean age in our study was 55 years ranging from 11-80 years. The mean age in our study was slightly higher in comparison to a study conducted in hospital of China by Shen et al[70] which may be due to delayed seeking of medical aid in our country. Jaundice was the commonest symptom. The most common auto antibodies found were ANA which were positive in 80% patients. This was in concordance with most other studies as conducted by Gurung et al[71] in which also ANA was positive in 98% cases while in the study done by Shen et al[72] showed LKM to be most common antibody.

Among the histological features lymphoplasmacytic infiltration in portal tracts was seen in all patients which is higher in number compared to

the other studies done by Gurung et al[71] and Shen et al[70] where it was present in around 60% patients. Rosetting and emperipolesis was seen in 9% of cases in our study. However, its incidence had varied significantly in the prior studies[31,72,73]. This is neither sensitive nor specific for diagnosis of autoimmune hepatitis which is also showed in a study done by Balitzer et al[74]

Primary biliary cirrhosis- We described a group of 8 patients of PBC in which mean age was 37 years having female predominance. Among laboratory manifestations, raised ALP was most common manifestation as was seen in study done by Drebber et al[32]. His study also took into consideration histological features like florid duct lesion and ductular reaction with periportal fibrosis. However, in our study patients with these findings were very less as compared to his study. Reason could be very large sample size of his study as compared to ours (N=232, n=8) Among histological patterns most common pattern was cholestasis which was also shown by Portmann et al[75] over 209 patients in which also 77% patients had similar pattern. AMA was positive in 100% patients in our study although AMA negative PBC can be seen in 5-10% cases[76].

Primary sclerosing cholangitis is the chronic cholestatic liver disease of unknown etiology. It is a disease of young males[77,78]. In the present study also M:F ratio was 2.5:1 and were in predominantly in the age group of 30-40 years. The predominant histological pattern was periductal fibrosis which was also shown by study done by Avino et al[79].

Overlap syndrome- In our study there were 3 cases with mean age of 37 years and all were females. In a study conducted by Chazouilleres et al[80] on 130 patients of which 12 cases had overlap syndrome, 10 patients were females as seen in our study. All had complained of jaundice. Ultrasonography was suggestive of cirrhosis of the liver with portal hypertension in 2 cases and chronic liver disease in 1 case. Laboratory tests showed raised AST and ALT levels, total bilirubin and ALP.

The AIH-PBC overlap syndrome is accepted when two or three criteria for PBC and AIH are fulfilled. In our cases for AIH - the criteria met included strong positivity for ANA and liver biopsy showing interface hepatitis. For PBC, the criteria met included strong positivity for AMA and liver biopsy showing bile duct lesions. For these reasons, overlap syndrome was diagnosed. Similar findings were given in a case report by Bairy et al[81] in which also a female of 50 years presented with jaundice and ANA and AMA positivity. Histological features met the criteria for overlap syndrome. Hence presence of autoantibodies, clinical and serological finding all favoured overlap syndrome.

Acute viral hepatitis–In our study there were total 10 cases of acute viral hepatitis of which 7 cases were anti HAV antibodies positive and 3 cases were anti HCV antibodies positive. There was male preponderance with M:F ratio of 2:1 and 60% patients were <40 years of age. Similar results were given by a study done by Agrawal et al[82] on 475 subjects where also most of the patients were males and were < 40 years of age. In the study conducted by Lenggenhager et al[83] portal inflammation was the most common histological feature (50%) followed by lobular hepatitis (21%). In our study lobular hepatitis was more common.

Chronic viral hepatitis - Of the total 366 cases, 64 cases (17.4%) had chronic viral hepatitis. Most of the patients were on treatment for CVH and liver biopsy was done to see for disease progression. This was in concordance with study done at Lagos[84] where chronic viral hepatitis was seen in 17.7% cases undergoing liver biopsies while few other studies as done by David et al[85] and Ugiagbe et al[86] showed much higher rates (55.2% and 62.5%) of all the patients undergoing liver biopsy. In our study males were affected predominantly with male to female ratio of 2.5:1. The age of the patients ranged from 17 to 75 years with peak in third decade of life. This may be explained by high risk behaviour of the young population. Among the liver function tests mean AST was 95 IU/L and mean ALT was 89 IU/L.AST> ALT was seen in 28 patients confirmed the finding of CVH. Mean bilirubin was 6 mg/dL and was raised in 26 cases. The CVH cases were reviewed on

the basis of modified Ishak score. 98% of the patients had minimal and mild grade disease with score of 1-3 and 4-8 respectively. This was in concordance with almost all the other studies as done by Desmet et al[87] where 70.5 % had similar findings. Another study done by Okafor et al[84] showed minimal disease in 46% of the patients and mild disease in 38% of the patients compared to our study which had 67% of minimal disease and 31% of mild grade. Similarly, a prospective study of 344 patients of CVH in Pakistan by Anwar et al[88] also showed majority of patients with minimal to mild disease.

Most of the patients in our study had fibrosis stage of 0-2 similar to the majority of studies while a study done by Ananya et al[89] reported majority of patients in stage 3.

Although several histological patterns have been described in patients with CVH of different aetiologies but none of them facilitates a definite histological diagnosis and the criteria to be described must be regarded as suggestive rather than diagnostic. Until now the most consistent feature of hepatitis C has been formation of lymphoid follicles with germinal centres in portal tracts. This may also be rarely found in chronic HBV infection and may not be present in many patients of HCV. In the present study also, it was only present in 1 case of the total 11 cases.

Tuberculosis – In our study there were 8 patients who had hepatic tuberculosis. Mean age was 53 years which was similar to the study conducted by Vilaichone et al[90] where mean age was 42 years. His study on 20 patients also concluded that majority patients had raised ALP levels similar to our study where 50% patients had raised ALP levels with highest value reaching upto 1167 IU/L. Similar variations in LFTs were also seen in various studies done by Hersch et al[91],Essop et al[92] and Sabharwal et al[93]. The most common histological feature in our study was same as described in study by Vilaichone et al[90] that was granulomas with negative ZN stain for acid fast bacilli.

Sarcoidosis is a multisystem inflammatory granulomatous disease of unknown etiology. In the present study mean age of the patients was 58 years with M: F of 1:1.2. This is in concordance with study done by

Ennaifer et al[94] while study done by Cremers et al[95] showed male predominance. All these studies done on hepatic sarcoidosis have very small number of patients. So large data-based study is required for these characteristics. Among abnormal LFTs most common seen was raised ALP revealing a cholestatic pattern. Bilirubin was raised in 22% patients. This cholestatic pattern seems to be due to progressive interlobular bile duct injury due to inflammatory infiltration of basement membrane and portal granuloma formation. It has been proposed that latter results in fibrosis of portal tracts and vanishing bile ducts. The occurrence of fibrosis was in 30% cases which was in concordance with findings of bridging fibrosis in 30% cases of previous studies.

Primary hepatic amyloidosis is characterised by abnormal deposition of monoclonal immunoglobulin light chains in liver. In the present study mean age was 58 years with male predominance similar to primary amyloidosis involving other sites. Among the laboratory tests elevation of ALP was the most common laboratory finding. This was in concordance with almost all studies done on hepatic amyloidosis like Gertz et al[96].

Among the histological pattern the predominant pattern was sinusoidal seen in 50% patients followed by mixed pattern involving both sinusoidal and portal tracts (40%) followed by vascular pattern (10%). Similar findings were also seen in a study done by Park et al[36] over 98 patients where also sinusoidal was predominant pattern seen in 66% patients.

Wilson disease- there was a single case of 28 years male who had presented with recurrent jaundice with raised serum bilirubin levels predominantly conjugated. SGOT were more than SGPT with ratio of 2.5 and this ratio of more than 2.2 has a sensitivity of 94% and a specificity of 86%[97]. Urinary copper levels of 24 hrs and serum ceruloplasmin levels were both raised.

On microscopy biopsy showed nodule formation separated by thick fibrous septae. Interface hepatitis was seen with intra-cellular and canalicular cholestasis. Rhodanine stain highlighted copper within the hepatocytes in the form of red cytoplasmic granules both in peri septal

area and in centrilobular hepatocytes. These findings were very similar to a case study reported by Gopal et al[103] where also young patient had jaundice and deranged LFTs with raised urinary 24 hrs copper with depressed ceruloplasmin levels. Liver biopsy had almost similar findings as in our case.

Gilbert syndrome- In our study there were 2 young males who had been suffering from a high rate of bile in the blood with normal clinical, laboratory and imaging examinations. Similar findings have been reported by Fikry et al[98]. Liver biopsy was normal except for presence of fine brown granular pigment in the hepatocytes- lipofuscin.

Dubin jonhson syndrome- In the present study there were 2 cases and both patients were in early adolescent age group but a study done by Cohen Y et al[99] has described that it can occur in neonatal age group also. Similar to the study done by Bangal et al[100] the presenting feature in both the patients were recurrent episodes of jaundice with high percentage of direct bilirubin: total bilirubin. Commonest histological diagnostic feature of coarse pigment was similar to all the cases.

Budd chiari syndrome- In our study there was single case of 45 years male. Ultrasonography revealed underlying veno occlusive disease. Total bilirubin was raised and other LFTs were normal. Liver biopsy revealed attempted nodule formation. Centrilobular vein showed perivenular fibrosis and dilated sinusoids around it and congestion of sinuisoids, focal dropout of centrilobular hepatocytes seen. Similar findings were explained by Simoes et al[101] in his case report.

BRIC/PFIC- A 30 years old male presented with jaundice, dark urine and pruritis for one month with mild hepatomegaly with normal bile ducts and no evidence of cirrhosis. Liver function tests revealed raised AST and ALT levels with normal ALP levels. Total and direct bilirubin was raised. These findings were similar to a case report of Chawla et al[102] of a 21-year-old young male with similar presentation.

Neonatal giant cell hepatitis- in our study there was a single case of 6 years female with complain of jaundice and ultrasonography revealed hepatomegaly. Total bilirubin was raised. Liver biopsy showed mainly

giant cell transformation. Portal tract inflammation was mild. Similar histological findings were explained by Torbenson et al[103] who did a study over the years in 2 tertiary centres. Giant cell change was the most common finding and mild to absent portal tract inflammation was seen in 97% cases.

Transplant rejection- in our study there was a 48 years male who underwent liver transplant due to cirrhosis and dysplastic nodule. Liver biopsy was received the same day of transplant. In our study RAI was 7 with global assessment rejection grade 3. There was study done Geramizadeh B et al[104] where out of 382 patients, 161 patients had acute rejection and 11.5% had severe rejection (RAI 7,8,9).

Obstructive cholestasis- In our study there were 14 cases where obstruction was the main pathology and bilirubin was raised in all cases. Various causes of obstructive cholestasis include inflammatory processes, choledocholithiasis, neoplasms like adenomas, papillomatosis, parasites, duodenal diverticulum, biliary atresia etc. in a study conducted by Morris et al[105] on 125 patients' various causes like gall stones in common duct, bile duct stricture, carcinoma head of pancreas etc have been explained.

SUMMARY& CONCLUSION

There were total 364 cases out of 11 cases had no specific pathology. Remaining 353 cases were analysed and divided on the base of aetiology.

- There were 97 cases (27.4%) of NAFLD. All the patients (100%) had some degree of fatty change either micro vesicular or macro vesicular. Majority patients had stage 4 fibrosis (30 cases, 30.9%) and 13 cases (13.4%) had no fibrosis.
- There were 22 cases (6.2%) were of ALD. Steatosis was seen in all the cases. Grade 1 ballooning degeneration was seen in 10 cases (45.5%). Majority patients had stage 4 fibrosis (13 cases, 59.2%).
- There were 13 cases of drug induced liver injury. Cholestatic pattern of injury was seen in all cases followed by necroinflammatory pattern in 9 cases (69.2%)
- Immune mediated hepatitis includes autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, overlap syndrome and transplant rejection. There were total of 71 cases in this category.
- 52 cases out of 71 cases were of autoimmune hepatitis. Lymphoplasmacytic portal inflammatory infiltrates were seen in all the cases. Majority patients (48%) had definite cirrhosis and only 5.8% patients had stage 1 fibrosis. 9.6% cases had no fibrosis.
- Primary biliary cirrhosis was seen in 8 cases. AMA positivity was seen in all cases. Florid duct lesion and ductular reaction was seen in few of the cases.
- There were 7 cases of primary sclerosing cholangitis. Of these, 3 cases had periductal fibrosis.
- 3 cases were of overlap syndrome having features of both autoimmune hepatitis and primary biliary cirrhosis.
- There were total 74 cases of viral hepatitis. Ten cases were of acute viral hepatitis and 64 cases were of chronic viral hepatitis.
- There were 7 cases of HAV and 3 of HEV. Lobular disarray along with spotty and confluent necrosis was seen in almost all cases.

- Amongst chronic viral hepatitis, 50 cases were caused by HBV infection and 14 cases were caused by HCV infection.
- HBsAg and HBV DNA were positive in all 50 cases of chronic hepatitis B and Anti HCV antibodies were positive in all 11 cases of chronic hepatitis C.
- Most of the patients of HBV had minimal chronic hepatitis (37 cases) and most of the patients of HCV had mild chronic hepatitis.
- Well formed, large epithelioid cell granulomas with lymphocytic collar were seen in 5 cases out of the 8 cases diagnosed as tuberculosis. Rest of the 3 cases had caseous necrosis, langhan's type giant cells and foreign body giant cells which gave the clue of tubercular aetiology.
- Sarcoidosis and amyloidosis were the infiltrative liver diseases included in my study.
- There were 10 cases of sarcoidosis and 8 cases of amyloidosis.
- Out of 10 cases of sarcoidosis, 5 cases had raised ACE levels. On microscopy, tiny multiple epithelioid cell granulomas were seen in the parenchyma. Granulomas were naked with no lymphocytic collar.
- There were 8 cases of amyloidosis of which sinusoidal pattern (linear) of amyloid deposition was the most common.
- Total of 15 cases lead to mechanical hepatic injury. Obstructive cholestasis was seen in 14 cases and 1 case was of vascular disorder- budd Chiari syndrome.
- There was a single case of Wilson's disease of a 28 years male who presented with jaundice. 24hrs urinary copper and ceruloplasmin levels were raised. Copper pigment was highlighted on Rhodanine stain.
- Hereditary defects of bilirubin included Gilbert's syndrome (2 cases), Dubin Johnson syndrome (2 cases) and BRIC/PFIC (1 case).
- There was single case of transplant rejection with global assessment rejection grade 3 (Severe).
- There were 17 cases of cirrhosis cause of which was unknown.

- Remaining cases included neonatal giant cell hepatitis (1 case), chronic hepatitis- idiopathic (6 cases) and cholestatic liver disease-idiopathic (5 cases).

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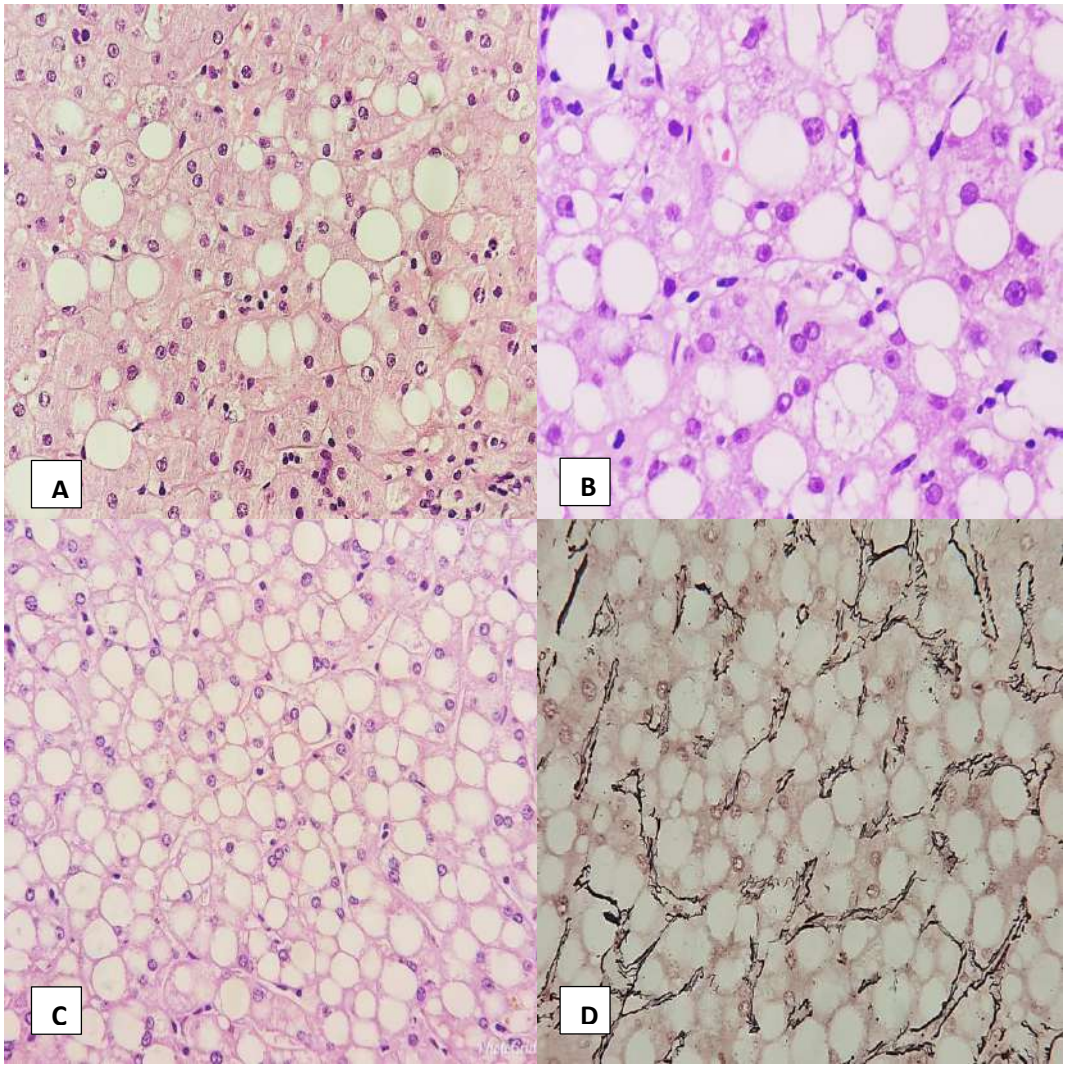
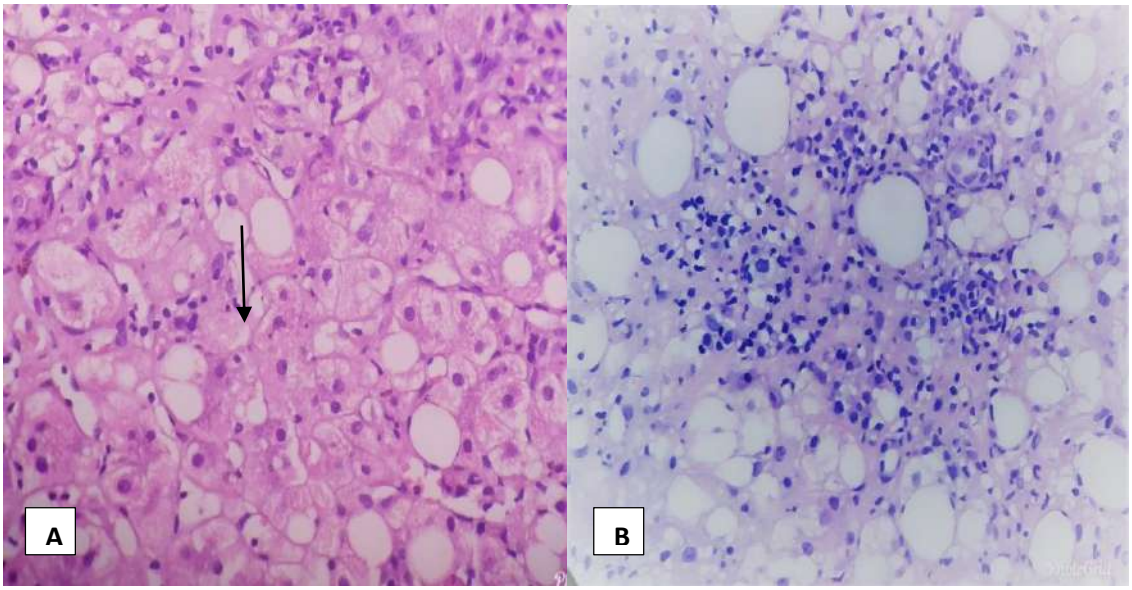
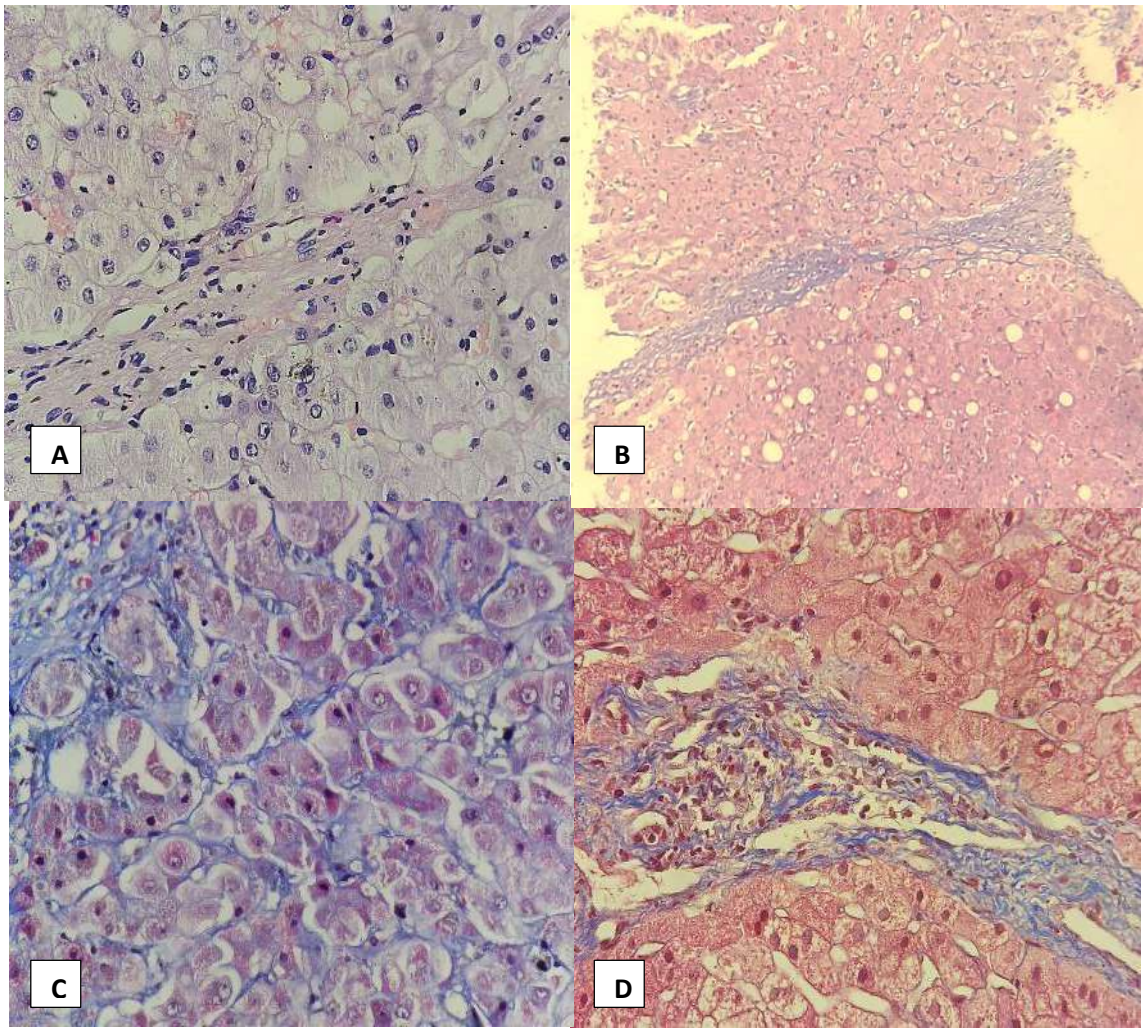


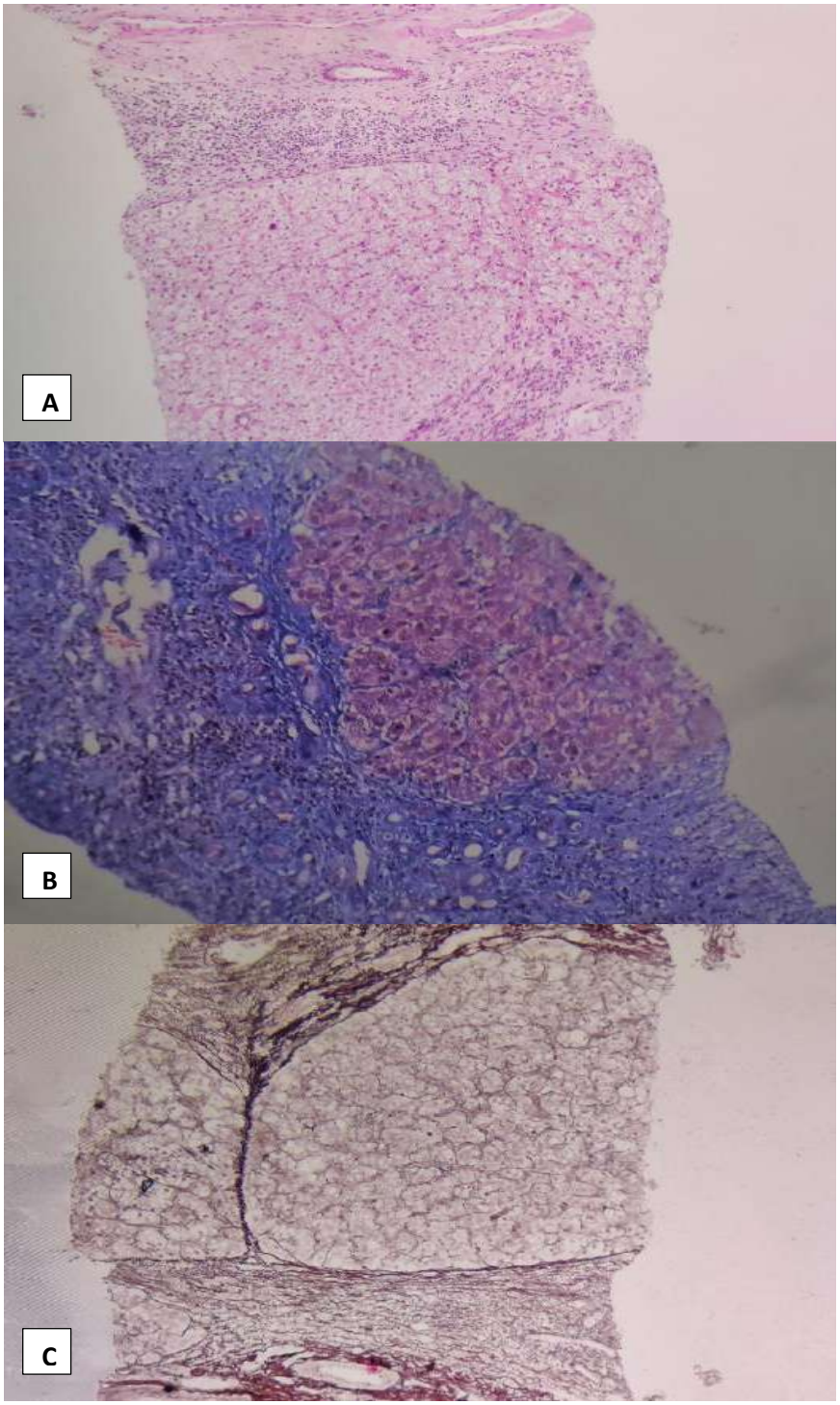
Picture 1: Photomicrograph of Non alcoholic fatty liver disease- Macrovesicular steatosis (A) Mild (5-10%) (B) Moderate (30-40%%) (C) Severe (>90%) (D) loss of reticulin in NASH (H&E, 400X)



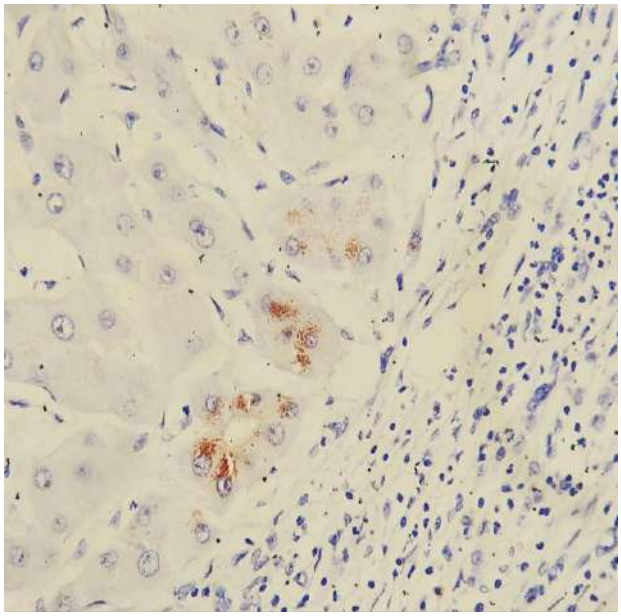
Picture 2: Photomicrograph of Non alcoholic steatohepatitis. (A) There is lobular infiltration by mixed inflammatory infiltrate – spotty necrosis. (B) Confluent necrosis (H&E, 400X)



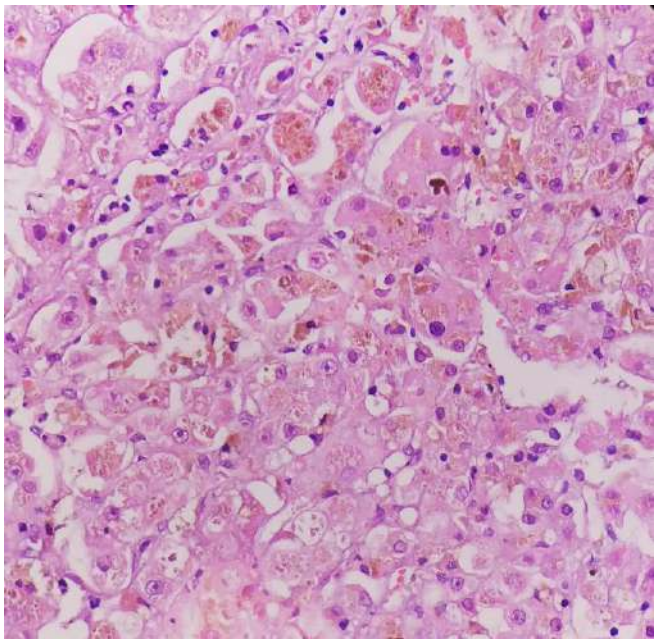
Picture 3 : Photomicrograph of NASH (A) Bridging fibrosis (H&E, 400X) (B) Bridging fibrosis (MT, 100X) (C) Pericellular fibrosis (MT, 400X) (D) Increased portal fibrosis with septa formation (MT stain, 400X)



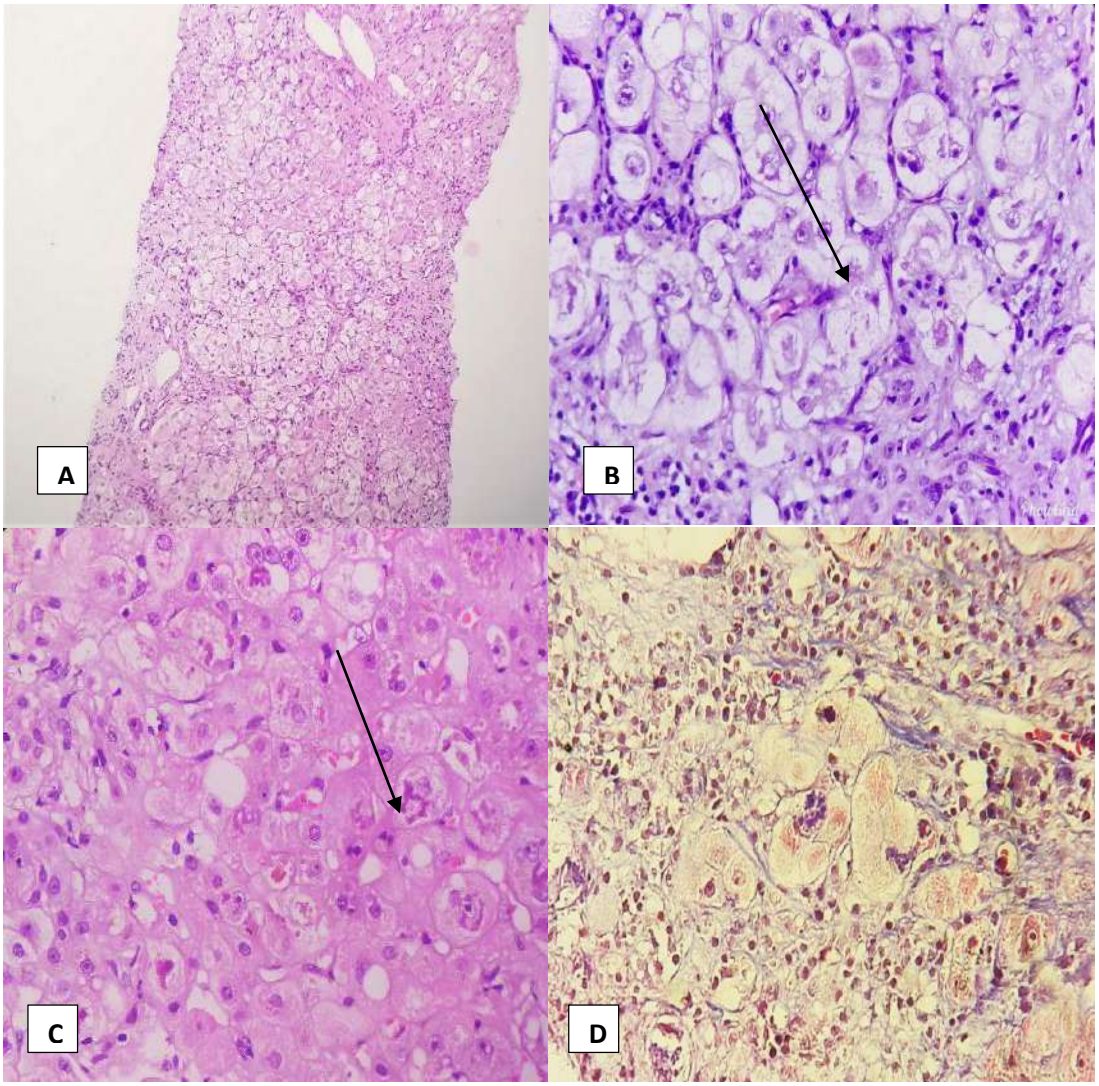
Picture 4: Photomicrograph of NASH leading on to cirrhosis exhibiting nodule formation (100X) (A) H&E stain (B) MT stain (C) Reticulin stain



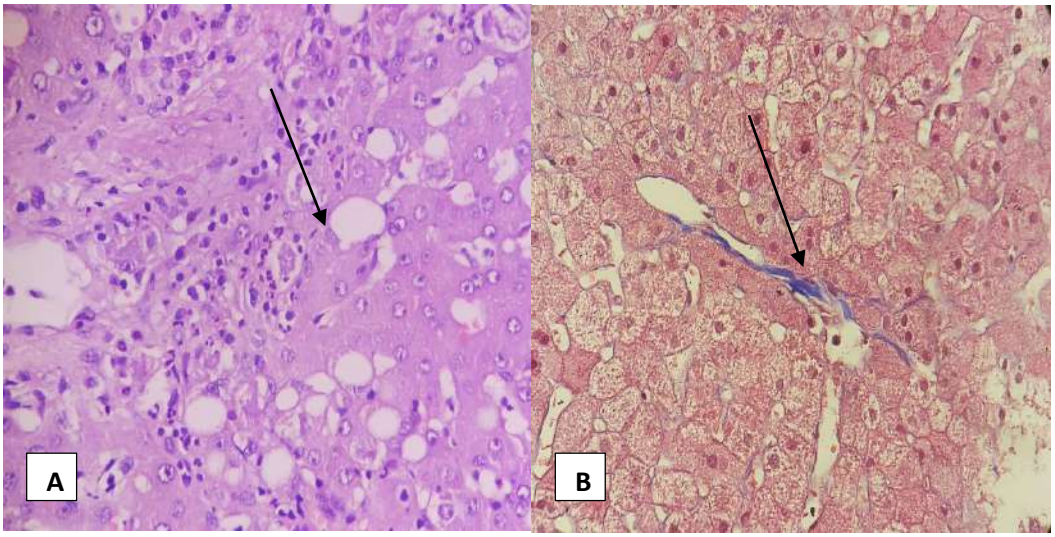
Picture 5 : Photomicrograph shows case of cirrhosis showing parenchymal deposition of copper pigmented highlighted on Rhodanine Stain (400X)



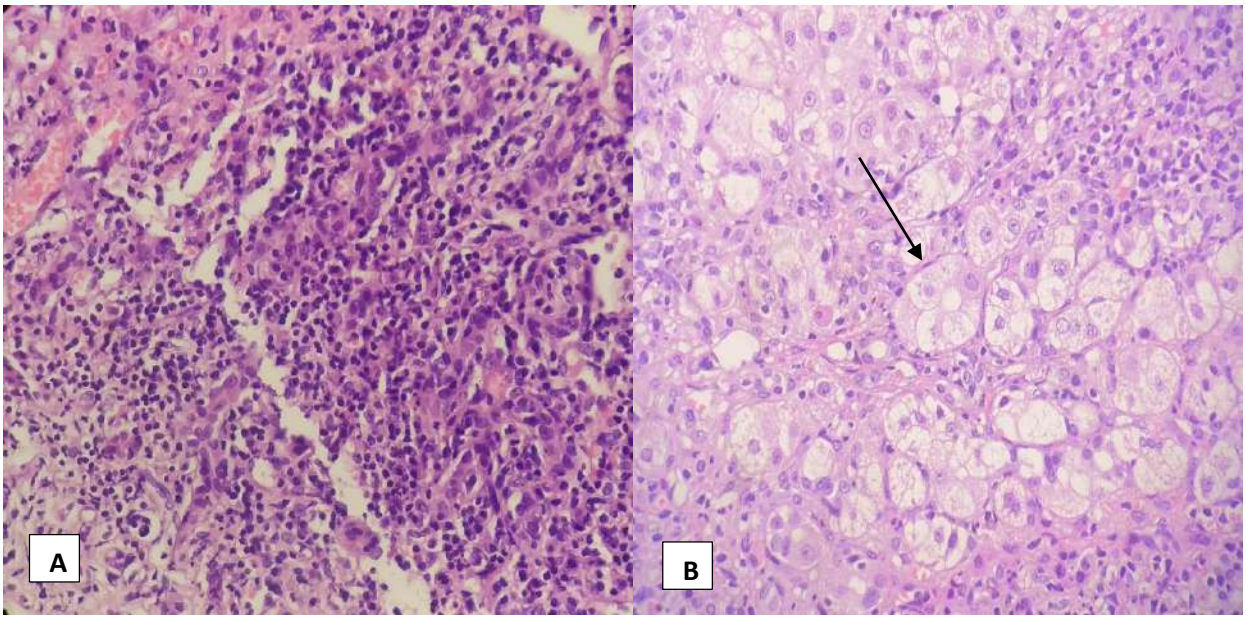
Picture 6 : Photomicrograph of case of Dubin Johnson syndrome showing diffuse coarsely granular brown pigment in liver cells (H&E, 400X)



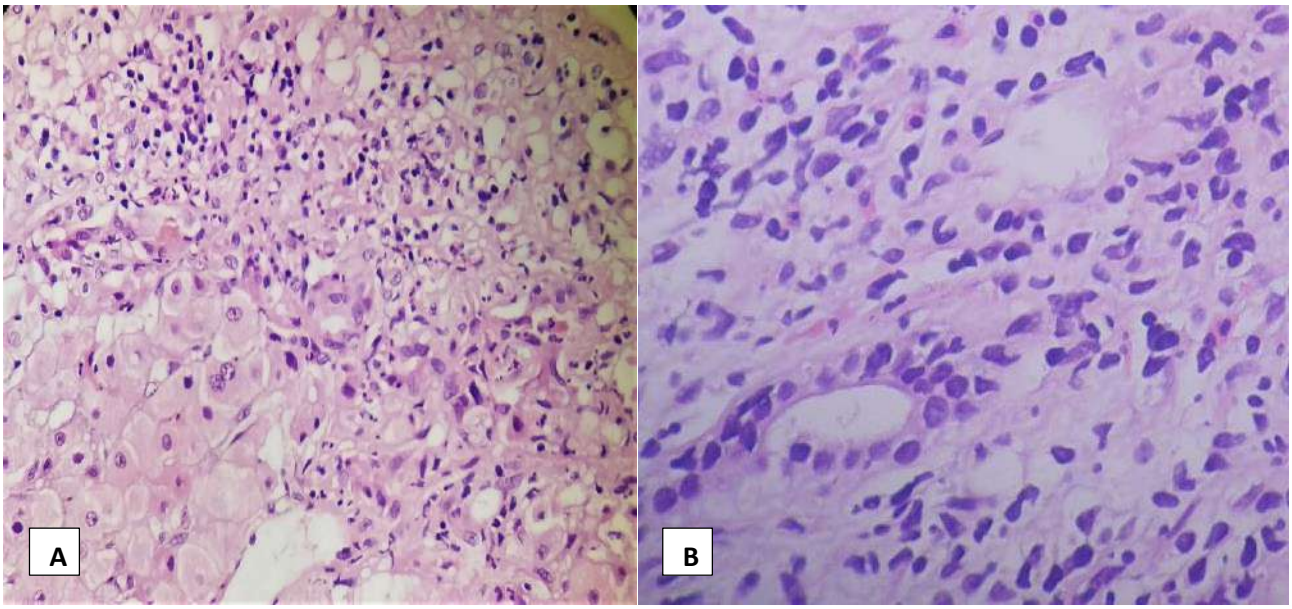
Picture 7: Photomicrograph of Alcoholic steatohepatits (A) Ballooning degeneration (H&E, 100X) (B) feathry degeneration (H&E, 400X) (C) Mallory denk bodies (H&E,400X) (D) Mallory Denk bodies (MT)



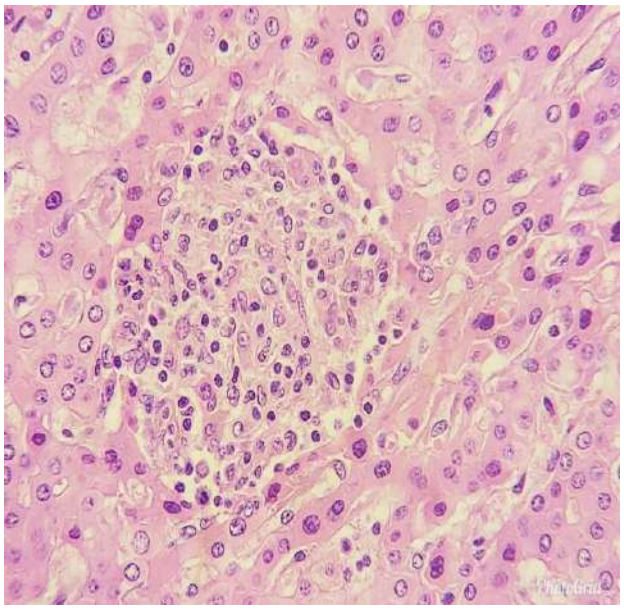
Picture 8 : Photomicrograph of Alcoholic steatohepatitis (A) Neutrophilic satellitosis (H&E, 400X) (B) Perivenular fibrosis (MT stain,400X)



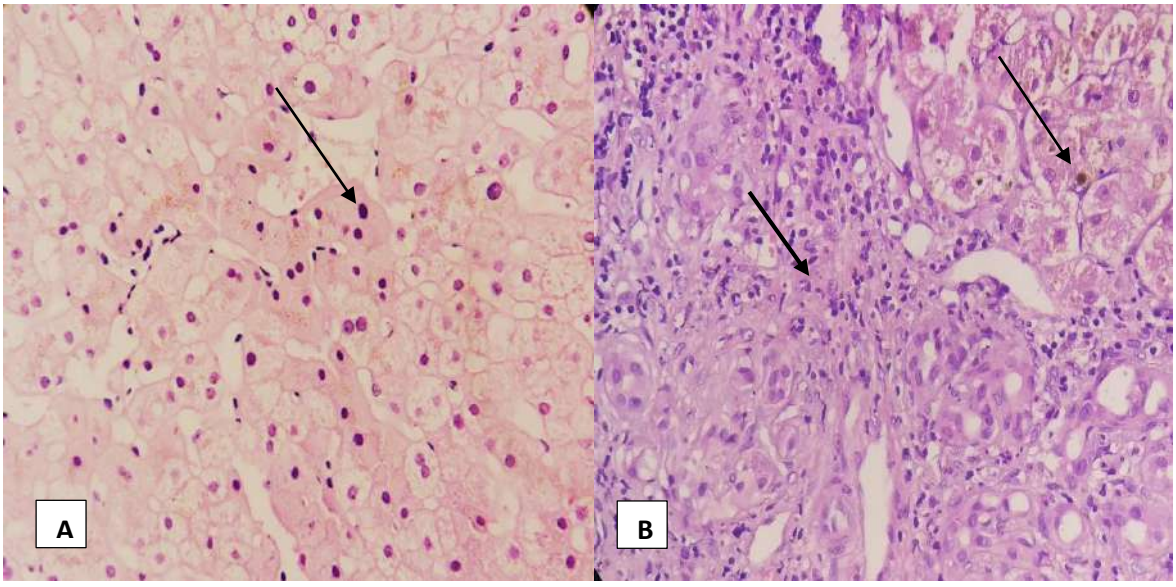
Picture 9 : Photomicrograph of autoimmune hepatitis (A) lobular infiltration by plasma cells (B) hepatocytic rosetting (H&E,400X)



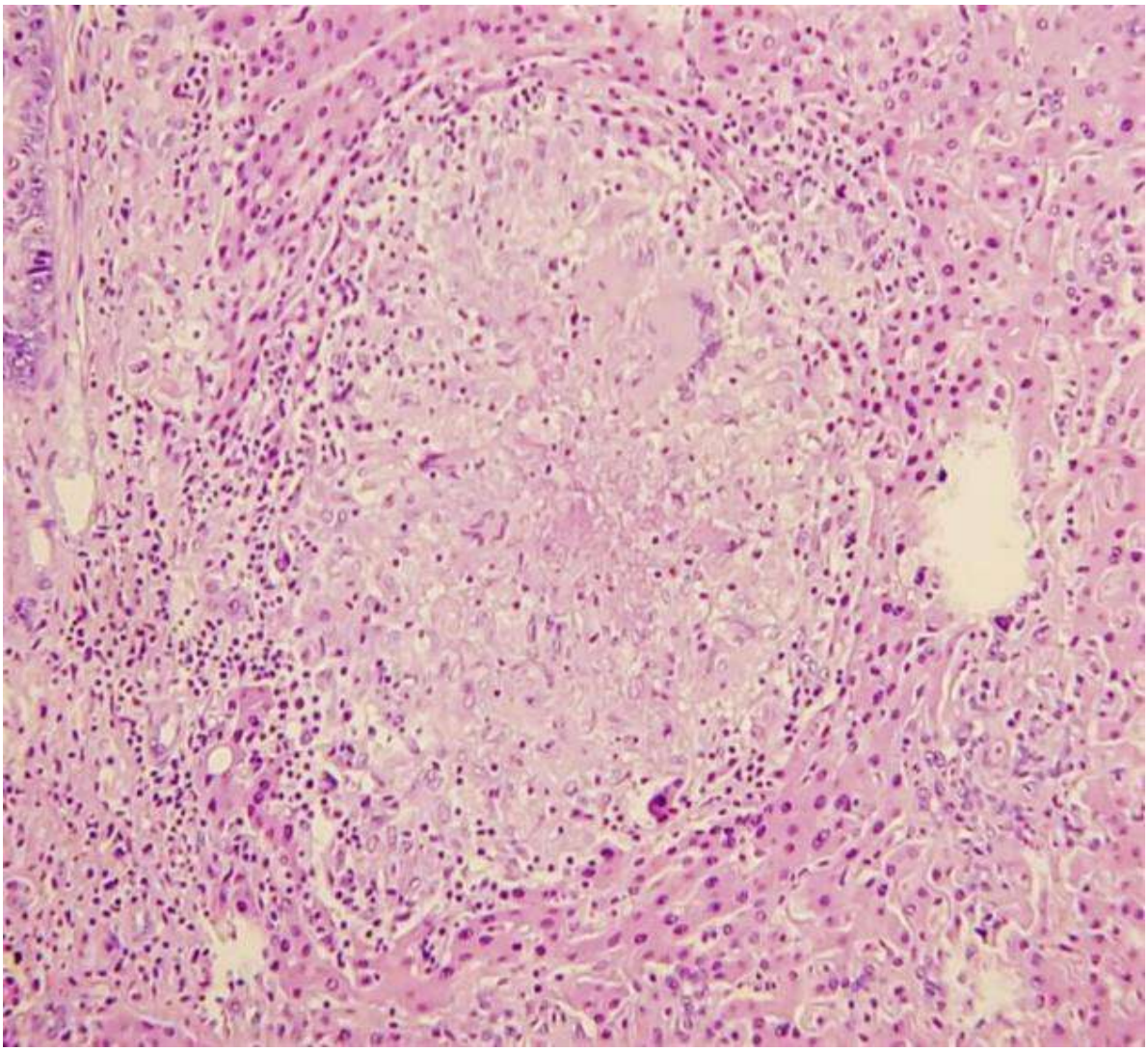
Picture 10 : (A) Photomicrograph of autoimmune hepatitis showing interface hepatitis (H&E, 400X) (B) Florid bile duct lesion (H&E, 400X)



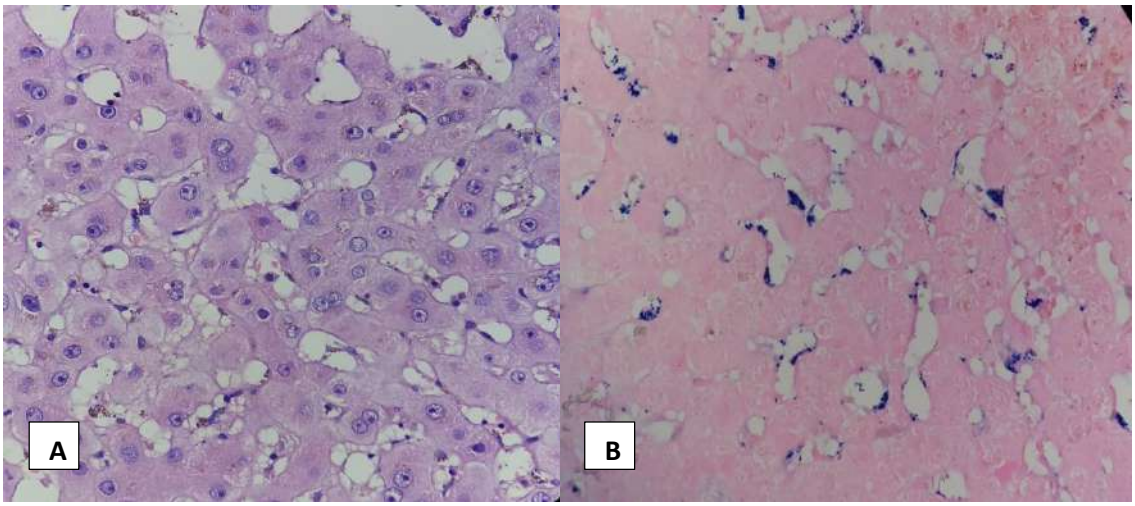
Picture 11 : Photomicrograph showing a case of primary biliary cirrhosis with granulomas (H&E, 400X)



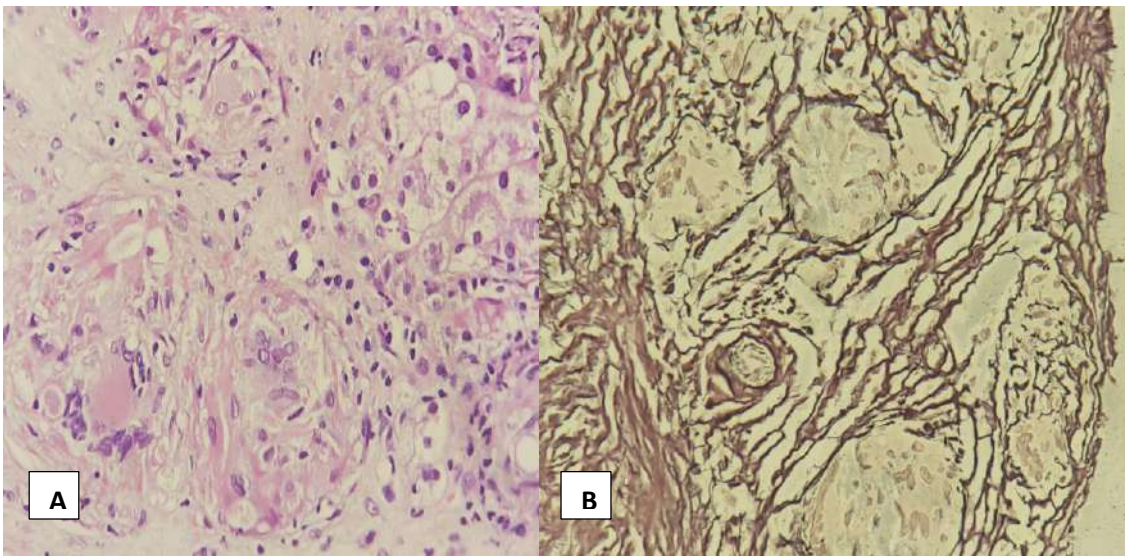
Picture 12 : Photomicrograph of Chronic viral hepatitis B (A) Ground glass hepatocytes (B) Bile duct proliferation (bold arrow) with lobular infiltrate and periportal intrahepatic cholestasis seen as bile plugs (H&E, 400X)



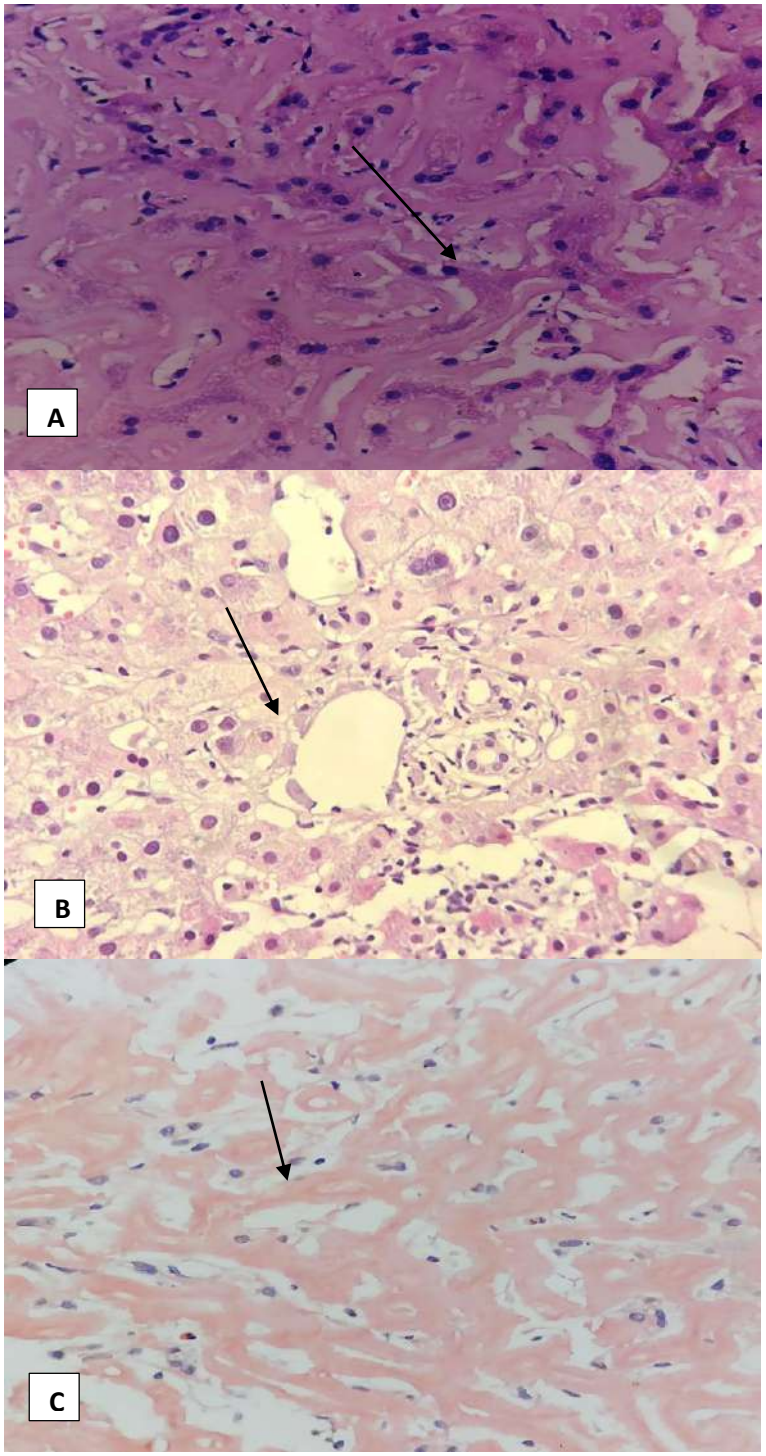
Picture 13: Photomicrograph showing tubercular granulomas with caseous necrosis (H&E, 400X)



Picture 14 : Photomicrograph of cirrhosis showing kupffer cell prominence with iron deposition (A) H&E stain (B) Perl's stain (400X).



Picture 15 : Photomicrograph of sarcoidosis (A) Show many small naked granulomas (H&E,400X) (B) Reticulin stain accentuating around the granulomas (400X)



Picture 16 : Photomicrograph of amyloidosis (A) linear pattern of amyloid deposition (H&E,400X) (B) section shows eosinophilic acellular globules around the central vein and in portal mesenchymal tissue (H&E, 400X) (C) Congo red stain highlighting amyloid(400X)

				HISTORY									BIOCHEMISTRY								
HISTO NO	INITIALS	MRD	AGE/SEX	JAUNDICE	DIABETES	ALCOHAL	OBESITY	ABDOMINAL MASS	HEPATO MEGALY	SPLENOME GALY	LYMPHADENO PATHY	RADIOLOGICAL FINDINGS	BILI TOTAL	BILI DIRECT	SGOT	SGPT	ALBUMIN	ALP	PROTIENS	VIRAL MARKERS	AUTOIMMUNE MARKERS
15-5619	IK	96097	47/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	3.5	3.2	60	65	5.2	162	6.8	NR	ND
15-6236	DS	212561	65/M	P	AB	AB	AB	AB	AB	AB	AB	ND	11.2	16	90	100	4.5	140	8	NR	ANA, AMA, ANCA 1+
15-6319	BS	116137	60/M	AB	P	P	P	AB	P	AB	AB	ND	1	2.5	45	50	5	140	8	HBV-POSITIVE	ND
15-6463	RC	26371	24/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	3	2.5	60	70	5.2	130	8	HBV-POSITIVE	ND
15-7197	D	129999	20/F	P	AB	AB	AB	AB	AB	AB	AB	ND	13.5	12	70	72	5.8	132	6	NR	AMA 3+, ANA 2+, ANCA 1+
15-7269	RS	135128	33/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.5	4.2	45	48	6	130	8.5	HEP-B POSITIVE	ND
15-7277	KS	129445	40/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.5	6	120	125	4	100	8.5	NR	NEGATIVE
15-7779	PS	141972	33/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISAESE	3.8	3.5	55	60	5.2	120	8.2	NR	AMA 3+, ANA 1+, ANCA 2+, LKM 1+
15-7854	RP	138299	68/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.2	4.8	55	60	4.2	140	6.5	NR	ND
15-7903	NS	148564	23/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	2.2	1.5	60	65	5	126	9.5	HEP-B POSITIVE	ND
15-7996	RK	146261	48/M	P	P	AB	P	AB	AB	AB	AB	CIRRHOSIS	8.5	9	72	65	5	130	8.5	NR	ANA 1+, ASMA 1+
15-8123	KG	155531	37/F	P	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISAESE	30	32	80	90	4.2	140	9.2	NR	AMA 3+, ANA 2+, ASMA 1+
15-8385	NS	161887	48/M	AB	AB	AB	AB	AB	P	P	AB	HEPATOSPEENOMEGALY	2	1	60	65	6.4	150	8.8	HEP-B POSITIVE	ND
15-8387	KS	161893	24/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	4	2.5	55	60	6	152	8	HEP-B POSITIVE	ND
15-8393	HS	162216	28/M	AB	AB	P	AB	AB	P	AB	AB	ND	4	2.5	70	80	6	180	9	HBV-POSITIVE	ND
15-8703	MS	121506	55/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.5	0.5	82	90	4.5	240	7.5	NR	ND
15-8959	GS	171750	37/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.2	1	120	135	5.5	350	5	NR	ND
15-9108	KK	172635	38/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	2.5	1.8	65	90	5.2	152	5.6	NR	ND
15-9150	SK	172928	22/M	P	AB	AB	AB	AB	AB	AB	AB	ND	5.6	5.2	150	158	5.6	220	6.2	NR	ND
15-9646	SK	76611	45/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	6.5	6.2	162	175	6.5	250	6	HBV-POSITIVE	ND
15-10166	GS	80642	23/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.6	5	125	130	4.5	200	5	HBV-POSITIVE	ND
15-10296	KR	201220	61/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	2	1.8	140	152	5.2	180	4.5	NR	ND
15-11489	MS	234288	61/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.8	1.5	90	110	4.8	110	5.2	NR	ND
15-11535	RM	234684	45/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	2.5	2.2	110	120	5	125	6.5	NR	ND
16-08	HK	259978	42/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.2	5	35	45	3.5	120	6.8	NR	NEGATIVE
16-154	AG	2030	55/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	50	55	3.5	400	9	HBV-POSITIVE	NEGATIVE
16-224	GS	269133	60/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	4	5.2	50	62	5	130	9	HCV-POSITIVE	ND
16-281	SK	222907	45/M	P	AB	AB	AB	AB	AB	AB	AB	ND	4.8	5.2	52	64	4	100	6.8	NR	ND
16-348	BS	3189	42/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.74	0.32	22	33	5.5	93	7.9	NR	NEGATIVE
16-687	BS	158535	64/M	P	P	AB	AB	AB	AB	AB	AB	ND	2	1.5	60	72	5	140	6.8	HBV-POSITIVE	ANA 2+
16-828	NS	4802	23/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	2	1.5	50	55	5.5	50	7	HBV-POSITIVE	ND
16-1094	MK	255146	24/F	AB	AB	AB	AB	AB	AB	P	AB	ND	3	2.2	30	35	5.5	150	7.5	HBV-POSITIVE	NEGATIVE
16-1166	KS	12410	20/M	P	AB	AB	AB	AB	P	P	AB	HEPATOSPLEENOMEGALY	25	20	50	60	6	180	8	HEV	ND
16-1365	SK	27531	39/F	P	AB	AB	AB	AB	AB	AB	AB	ND	36.2	35	50	55	5	135	6.8	NR	ANA 2+ AMA 3+ LKM 1+
16-1487	KK	32079	37/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.2	1	42	50	5	80	8	HCV-POSITIVE	NEGATIVE
16-1878	BS	39603	62/M	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	0.86	0.26	35	22	4.8	75	8.5	NR	NEGATIVE
16-2098	RS	43093	27/F	P	AB	AB	AB	AB	P	AB	AB	CHOLESTATIC LIVER DISEASE WITH HEPATOMEGALY	15.3	12.2	45	52	5	140	8.8	NR	ANA 1+, SMA 1+
16-2191	RR	50634	58/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	2	1.5	50	55	4	140	8.2	NR	NEGATIVE
16-2355	JS	95109	17/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	3.5	2.2	36	42	4	130	7.2	HBV-POSITIVE	NEGATIVE
16-2413	SS	45868	22/M	P	AB	AB	AB	AB	AB	AB	AB	ND	18.2	15	52	55	4.2	60	7.2	HBV-POSITIVE	NEGATIVE
16-2543	SM	264399	52/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	2.2	1.5	50	55	5.5	150	7.2	HBV POSITIVE	NEGATIVE
16-2749	PG	23447	34/M	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	5.8	5	52	60	6	180	6.8	HBV-POSITIVE	ND
16-2931	JR	180900	50/M	P	AB	AB	P	AB	AB	AB	AB	ND	6.2	5.2	52	60	5.1	56	8	HBV-POSITIVE	NEGATIVE
16-3274	GS	52400	25/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	3.6	2.2	40	45	5	180	6.2	HBV-POSITIVE	NEGATIVE
16-3321	SS	126084	37/M	P	AB	AB	AB	AB	AB	AB	AB	ND	7.2	6.5	50	60	5.5	150	7.2	HBV-POSITIVE	ND
16-3466	JD	54294	62/F	AB	AB	AB	AB	AB	P	AB	AB	HEPATOMEGALY	16.5	15	60	70	5.5	200	8.2	NR	ANA 3+ ASMA3+, AMA3+, LKM 3+
16-3484	BLS	153753	57/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	12.2	10.5	60	70	4.5	140	6.8	NR	ND
16-3654	SK	81721	29/M	AB	AB	AB	AB	AB	P	P	AB	HEPATOSPLEENOMEGALY	1	0.2	35	8	3.5	120	6.8	NR	ND
16-3746	PS	54977	22/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.79	1.55	23	43	5.5	340	3.1	HBV-POSITIVE	NEGATIVE
16-3753	VS	79754	19/M	P	AB	AB	AB	AB	P	AB	AB	ND	15.3	12.2	50	65	5	180	7.2	NR	ND
16-3991	TS	250217	22/M	AB	P	AB	AB	AB	P	P	AB	HEPATOMEGALY	1.2	0.5	50	52	5	130	8	HBV-POSITIVE	ND
16-4146	JS	84260	22/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.2	0.12	19	24	4.34	107	7.7	HBV-POSITIVE	ND
16-4694	JS	98352	38/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.38	0.11	19	24	4.4	101	7.8	NR	ND
16-5056	SS	64606	26/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	6.2	5.2	40	45	4	120	7	HBV-POSITIVE	NEGATIVE
16-5106	SK	107065	52/F	AB	AB	AB	AB	AB	P	AB	AB	ND	3.6	2.2	55	60	4	130	8	NR	ND
16-5731	SS	91081	28/M	P	AB	AB	AB	AB	AB	AB	AB	ND	12.5	10.5	60	70	5.2	190	7.8	HBV-POSITIVE	ND
16-5951	RK	10858	44/M	P	AB	AB	AB	AB	AB	AB	AB	CHOLESTATIC LIVER DISEASE	25.5	20	50	60	5	250	8	NR	ND
16-5972	RB	116394	31/F	AB	P	AB	P	AB	AB	AB	AB	ND	3.6	2.2	55	60	5.2	150	9	HBV POSITIVE	NEGATIVE
16-6290	ADG	118379	70/F	AB	AB	AB	AB	AB	P	AB	AB	ND	15.2	12.5	36	50	4.2	250	7.5	HBV-POSITIVE	NEGATIVE
16-6352	GS	119315	22/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	8.4	7.2	70	75	2.5	250	7	HBV-POSITIVE	ND
16-6411	JS	119661	31/M	AB	AB	AB	AB	AB	P	AB	AB	ND	5	4.2	50	55	4.8	120	7.5	HBV-POSITIVE	NEGATIVE
16-6473	AS	117577	38/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5	4.2	50	55	4.5	200	8.2	HCV-POSITIVE	ND
16-6575	PK	120249	60/F	P	P	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	20.2	15.5	100	110	5.2	200	6.8	NR	ANA 3+, AMA 2+, LKM 2+, ASMA 3+
16-6612	BK	119870	60/F	P	AB	AB	AB	AB	P	P	AB	HEPATOSPLEENOMEGALY	30	26	40	45	4	300	5.2	NR	NEGATIVE
16-6780	MK	120947	43/F	AB	AB	AB	AB	AB	AB	AB	P	ND	2.4	1.2	50	60	5	160	6.9	NR	ND
16-7108	PR	123641	53/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	11.1	10.2	60	75	5	200	8	HBV-POSITIVE	ND
16-7744	SP	123593	42/F	P	AB	AB	AB	AB	P	AB	AB	CHRONIC LIVER DISEASE	9.6	8.2	40	45	5	110	8	NR	ANA 3+, ASMA 3+, LKM 1+
16-7810	GS	128887	16/M	P	AB	AB	AB	AB	AB	AB	AB	ND	12.5	10.2	50	55	5.5	200	6.2	NR	ANA 3+, AMA 2+, ASMA 2+, LKM 1+
16-7858	JS	128867	15/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	6.5	5.2	35	38	3.5	150	6.8	HBV-POSITIVE	NEGATIVE
16-7943	SB	129802	50/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.5	0.2	35	45	3.5	120	6.8	NR	ND
16-7964	JK	129816	39/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.6	4.2	60	64	4.5	142	5.2	HBV-POSITIVE	ND
16-7999	PK	130165	44/M	P	AB	AB	AB	AB	P	AB	AB	ND	9.6	8.2	70	75	6.2	250	7.5	NR	AMA3+, ANA2+, SMA3+,LKM 3+
16-8330	HK	132952	62/F	AB	P	AB	AB	AB	AB	AB	AB	ND	0.5	0.2	35	45	3.5	120	6.8	NR	ND
16-8313	KC	131725	40/M	AB	AB	AB	AB	AB	AB	AB	AB	CHOLESTATIC LIVER DISEASE	18.2	15.5	50	55	4	200	6	HEV- POSITIVE	ND
16-8476	NM	128420	24/M	P	AB	AB	AB	AB	AB	AB	AB	CHOLESTATIC LIVER DISEASE	20.5	15.2	110	115	4	300	6.8	HAV-POSITIVE	ND
16-8827	RK	136175	40/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	6	5.2	100	110	5	210	8.2	HBV-POSITIVE	ND
16-9096	G	130467	1MONTH/M	P	AB	AB	AB	AB	AB	AB	AB	ND	35.5	30.2	70	75	5.1	130	8	NR	ND
16-9234	CK	139411	42/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	35	45	3.5	120	6.8	NR	NEGATIVE

HISTO NO	INITIALS	CERULOPLASMIN	IRON	LIVER BIOPSY GROSS NUMBER	LENGTH	ARCHITECTURE	FATTY CHANGE TYPE	FATTY CHANGE DEGREE
15-5619	IK	ND	ND	1	1.5CM	MAINTAINED	AB	AB
15-6236	DS	15	ND	1	1.2 CM	MAINTAINED ARCHITECTURE	AB	AB
15-6319	BS	20	ND	1	1.5 CM	DISTORTED WITH NODULE FORMATION	AB	AB
15-6463	RC	40	ND	1	1.5 CM	MAINTAINED	AB	AB
15-7197	D	ND	ND	2	0.5 & 1.0	MAINTAINED	AB	AB
15-7269	RS	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
15-7277	KS	26	ND	2	0.8 & 1.5 CM	DISARRAY	MACROVESICULAR	0.3
15-7779	PS	ND	ND	2	1.0 & 1.2 CM	DISTORTED WITH NODULE FORMATION	AB	AB
15-7854	RP	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
15-7903	NS	ND	ND	1	1.5CM	MAINTAINED	MACROVESICULAR	0.4
15-7996	RK	20	ND	2	1.0 & 1.2 CM	DISTOTED WITH BROAD PORTAL SCAR	MACROVESICULAR	0.2
15-8123	KG	25	ND	1	0.8 CM	DISARRAY	AB	AB
15-8385	NS	ND	ND	2	0.6 & 0.8 CM	MAINTAINED	AB	AB
15-8387	KS	ND	ND	1	1.2 CM	MAINTAINED	MACROVESICULAR	0.1
15-8393	HS	25	ND	1	1.0 CM	MAINTAINED	MACROVESICULAR	0.05
15-8703	MS	ND	ND	1	1 CM	MAINTAINED	AB	AB
15-8959	GS	ND	ND	1	1.2CM	LOBULAR DISARRAY	MACROVESICULAR	0.7
15-9108	KK	ND	ND	2	1 & 1.5 CM	MAINTAINED	MACROVESICULAR	0.1
15-9150	SK	ND	ND	1	1.5 CM	MAINTAINED	AB	AB
15-9646	SK	ND	ND	1	1 CM	MAINTAINED	AB	AB
15-10166	GS	ND	ND	1	1.8CM	MAINTAINED	AB	AB
15-10296	KR	ND	ND	1	1.3 CM	DISARRAY	MACROVESICULAR	0.15
15-11489	MS	ND	ND	2	0.8 & 1 CM	MAINTAINED	MACROVESICULAR	0.8
15-11535	RM	ND	ND	1	1.5 CM	MAINTAINED	MACROVESICULAR	0.66
16-08	HK	ND	ND	1	1.2 CM	MAINTAINED ARCHITECTURE	AB	AB
16-154	AG	ND	ND	2	1.0 & 1.5 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	0.05
16-224	GS	ND	ND	2	0.6 & 0.8 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-281	SK	ND	ND	1	1.2 CM	DISTORTED ARCHITECTURE LARGE SCARED AREAS	AB	AB
16-348	BS	ND	ND	1	1.2 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.05
16-687	BS	35	ND	2	0.8 & 0.6 CM	MAINTAINED ARCHITECTURE	AB	AB
16-828	NS	ND	ND	1	1.8 CM	MAINTAINED LOBULAR AND PORTAL ARCHITECTURE	AB	AB
16-1094	MK	ND	ND	2	1.5 & 1 CM	MAINTAINED ARCHITECTURE	AB	AB
16-1166	KS	18	ND	1	0.8 CM	LOBULAR DISARRAY	AB	AB
16-1365	SK	ND	ND	1	1.2 CM	DISTORTED ARCHITECTURE	AB	AB
16-1487	KK	ND	ND	2	0.6 & 0.8 CM	LOBULAR DISARRAY	MACROVESICULAR	0.1
16-1878	BS	22	ND	1	1.2 CM	DISTORTED WITH LOBULAR FORMATION	AB	AB
16-2098	RS	22	ND	2	0.8 & 1.2 CM	DISTORTED WITH NODULE FORMATION	AB	AB
16-2191	RR	ND	ND	1	1.5 CM	DISTORTED HEPATIC ARCHITECTURE WITH BROAD PORTAL SEAR	MACROVESICULAR	0.2
16-2355	JS	ND	ND	2	0.6 & 1 CM	MAINTAINED LOBULAR ARCHITECTURE	AB	AB
16-2413	SS	ND	ND	1	1.6CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-2543	SM	26	ND	1	1.5CM	MAINTAINED ARCHITECTURE	AB	AB
16-2749	PG	ND	ND	1	1.2 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.1
16-2931	JR	ND	ND	2	0.8 & 1.2 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	MACROVESICULAR	0.05
16-3274	GS	ND	ND	1	0.8 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
16-3321	SS	ND	ND	2	0.6 & 0.8 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	0.2
16-3466	JD	ND	ND	2	0.8 & 1.0 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-3484	BLS	ND	ND	2	1 & 1.2 CM	DISTORTED WITH NODULE FORMATION	AB	AB
16-3654	SK	ND	ND	1	1 CM	DISTORTED ARCHITECTURE	MACROVESICULAR	0.66
16-3746	PS	ND	45.6	1	1.2 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	0.05
16-3753	VS	35	ND	1	1.2 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION	AB	AB
16-3991	TS	22	70	2	1.2 & 1.5 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	0.4
16-4146	JS	ND	ND	1	1.5 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
16-4694	JS	ND	ND	1	1.0 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	MACROVESICULAR	AB
16-5056	SS	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
16-5106	SK	25	ND	1	1 CM	MARKED DISTORTION WITH NODULE FORMATION	AB	AB
16-5731	SS	ND	ND	1	1 CM	MAINTAINED ARCHITECTURE	AB	AB
16-5951	RK	ND	ND	1	0.6 CM	DISARRAY WITH ATTEMPTED NODULE FORMATION	AB	AB
16-5972	RB	20	60	1	2 CM	MILD DISARRAY	AB	AB
16-6290	ADG	ND	ND	1	2 CM	MAINTAINED ARCHITECTURE	AB	AB
16-6352	GS	ND	ND	2	0.4 & 0.6 CM	MAINTAINED ARCHITECTURE	AB	AB
16-6411	JS	42	60	1	1 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	AB
16-6473	AS	ND	ND	1	1 CM	MAINTAINED ARCHITECTURE	AB	AB
16-6575	PK	ND	ND	1	1 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-6612	BK	ND	ND	2	0.2 & 0.8 CM	MAINTAINED ARCHITECTURE	AB	AB
16-6780	MK	ND	ND	2	1.2 & 1.5 CM	MAINTAINED ARCHITECTURE	AB	AB
16-7108	PR	ND	ND	3	0.3, 0.4 & 0.6 CM	MILD DISSARRAY	MACROVESICULAR	AB
16-7744	SP	ND	ND	1	1 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
16-7810	GS	ND	ND	1	1.2 CM	DISARRAY OF PORTAL AND LOBULAR ARCHITECTURE	AB	AB
16-7858	JS	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
16-7943	SB	ND	ND	1	0.7 CM	MAINTAINED ARCITECTURE	AB	AB
16-7964	JK	26	72	1	1.5 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
16-7999	PK	ND	ND	1	0.8 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-8330	HK	ND	ND	1	1.2 CM	DISTORTED LOBULAR ARCHITECTURE WITH NODULE FORMATION	AB	AB
16-8313	KC	ND	ND	1	1 CM	MAINTAINED ARCHITECTURE	AB	AB
16-8476	NM	ND	ND	1	1.8CM	MAINTAINED ARCHITECTURE	AB	AB
16-8827	RK	ND	ND	1	0.9 CM	MAINTAINED ARCHITECTURE	AB	AB
16-9096	G	ND	ND	2	1.5 & 1.8 CM	DISARRAY	FOCAL STEATOSIS	AB
16-9234	CK	ND	ND	1	1.5 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.05

HISTO NO	INITIALS	CHOLESTASIS	BILE	BILE DUCT	MALLORY DENK BODIES
15-5619	IK	AB	AB	AB	AB
15-6236	DS	PROMINENT	INTRAHEPATIC	BLE DUCT PROLIFERATION	AB
15-6319	BS	AB	AB	AB	AB
15-6463	RC	AB	AB	AB	AB
15-7197	D	P	AB	DUCTULAR PROLIFERATION NOTED	AB
15-7269	RS	AB	AB	AB	AB
15-7277	KS	AB	AB	AB	AB
15-7779	PS	AB	AB	AB	AB
15-7854	RP	AB	AB	AB	AB
15-7903	NS	AB	AB	AB	AB
15-7996	RK	INTRAHEPATIC AND INTRA CANALICULAR	BILE PLUGS	AB	P
15-8123	KG	P	BILE PLUGS	AB	AB
15-8385	NS	AB	AB	AB	AB
15-8387	KS	AB	AB	AB	AB
15-8393	HS	AB	AB	AB	AB
15-8703	MS	PRESENT	AB	AB	AB
15-8959	GS	PRESENT	AB	AB	P
15-9108	KK	AB	AB	AB	AB
15-9150	SK	AB	AB	AB	AB
15-9646	SK	AB	AB	AB	AB
15-10166	GS	AB	AB	AB	AB
15-10296	KR	AB	AB	AB	AB
15-11489	MS	AB	AB	AB	AB
15-11535	RM	INTRAHEPATIC AND CANALICULAR TYPE	AB	AB	AB
16-08	HK	AB	BILE PLUGS	REACTION	AB
16-154	AG	P	AB	AB	AB
16-224	GS	INTRACELLULAR CHOLESTASIS	STASIS	AB	AB
16-281	SK	AB	STASIS	AB	AB
16-348	BS	INTRACELLULAR CHOLESTASIS	AB	AB	AB
16-687	BS	AB	AB	AB	AB
16-828	NS	AB	AB	AB	AB
16-1094	MK	AB	AB	AB	AB
16-1166	KS	INTRACELLULAR BILIRUBINESTASIS	BILE PLUGS	AB	AB
16-1365	SK	INTRAHEPATIC CHOLESTASIS	STASIS	AB	AB
16-1487	KK	AB	AB	AB	AB
16-1878	BS	AB	AB	AB	AB
16-2098	RS	P	BILE PLUGS	REACTION	AB
16-2191	RR	AB	AB	AB	AB
16-2355	JS	AB	AB	AB	AB
16-2413	SS	AB	AB	AB	AB
16-2543	SM	AB	AB	REACTION	AB
16-2749	PG	AB	AB	AB	AB
16-2931	JR	AB	AB	AB	AB
16-3274	GS	AB	AB	AB	AB
16-3321	SS	AB	AB	AB	AB
16-3466	JD	AB	AB	AB	AB
16-3484	BLS	AB	BILIRUBINESTASIS	AB	AB
16-3654	SK	AB	AB	AB	AB
16-3746	PS	AB	AB	AB	AB
16-3753	VS	INTRACANALICULAR	AB	REACTION	AB
16-3991	TS	AB	AB	AB	AB
16-4146	JS	AB	AB	AB	AB
16-4694	JS	AB	AB	AB	AB
16-5056	SS	AB	AB	AB	AB
16-5106	SK	AB	AB	AB	AB
16-5731	SS	AB	AB	AB	AB
16-5951	RK	AB	AB	P	AB
16-5972	RB	AB	AB	AB	AB
16-6290	ADG	AB	AB	AB	AB
16-6352	GS	AB	AB	AB	AB
16-6411	JS	AB	AB	AB	AB
16-6473	AS	AB	AB	AB	AB
16-6575	PK	AB	AB	AB	AB
16-6612	BK	AB	AB	AB	AB
16-6780	MK	AB	AB	AB	AB
16-7108	PR	AB	AB	AB	AB
16-7744	SP	AB	AB	P	AB
16-7810	GS	AB	AB	AB	AB
16-7858	JS	AB	AB	AB	AB
16-7943	SB	AB	AB	AB	AB
16-7964	JK	AB	AB	AB	AB
16-7999	PK	AB	AB	REACTION	AB
16-8330	HK	AB	AB	AB	AB
16-8313	KC	P	BILE PLUGS	AB	AB
16-8476	NM	P	P	AB	AB
16-8827	RK	AB	AB	AB	AB
16-9096	G	AB	AB	AB	AB
16-9234	CK	AB	AB	AB	AB

HISTO NO	INITIALS	HEPATOCYTES
15-5619	IK	EOSINOPHILIC ACELLULAR EXTRACELLULAR MATERIAL
15-6236	DS	MILD BALLONING DEGENERATION
15-6319	BS	MILD BALLOING DEGENRATION
15-6463	RC	GROUND GLASS HEPATOCYTES NOTED
15-7197	D	MILD BALLOING DEGENRATION
15-7269	RS	MILD BALLONIG DEGENRATION
15-7277	KS	NON NECROTIZING GRANULOMAS WITHOUT ANY LYMPHOCYTIC COLLAR. OCCASIONAL GIANT CELLS SEEN.
15-7779	PS	MILD BALLONIG DEGENRATION
15-7854	RP	MARKED PERISINUSOIDAL WALL WITH PINKISH HYALINE HOMOGENEOUS ACELLULAR MATERIAL- AMYLOID. CONGO RED STAIN IS POSITIVE AND APPLE GREEN
15-7903	NS	MILD BALLONIG DEGENRATION
15-7996	RK	MILD BALLOONING DEGENRATION
15-8123	KG	MILD BALLONIG DEGENRATION
15-8385	NS	GROUND GLASS HEPATOCYTES NOTED
15-8387	KS	GROUND GLASS HEPATOCYTES NOTED
15-8393	HS	GROUND GLASS HEPATOCYTES NOTED
15-8703	MS	ROSETTING NOTED
15-8959	GS	MILD BALLOONING
15-9108	KK	MODERATE BALLOONING DEGENERTION
15-9150	SK	ABUNDANT CAUSE DARK BROWN KYPOPLASMIC PIGMENT
15-9646	SK	BALLOONING DEGENERATION
15-10166	GS	BALLOONING DEGENERATION
15-10296	KR	MODERATE BALLOONING DEGENERTION
15-11489	MS	MILD BALLONING DEGENERATION
15-11535	RM	MODERATE BALLOONING DEGENRATION
16-08	HK	MILD BALLONIG DEGENRATION
16-154	AG	NP
16-224	GS	NP
16-281	SK	MILD BALLONIG DEGENRATION
16-348	BS	UR
16-687	BS	NP
16-828	NS	NP
16-1094	MK	GROUND GLASS APPEARANCE NOTED
16-1166	KS	FEATHRY DEGENERATION MILD BALLOONING DEGENERATION
16-1365	SK	BALLOONING AND FEATHRY DEGENERATION
16-1487	KK	BALLOONING DEGENERATION
16-1878	BS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-2098	RS	ROSETTE FORMATION AND GIANT CELL TRANSFORMATION
16-2191	RR	MILD BALLONING DEGENERATION
16-2355	JS	NP
16-2413	SS	NP
16-2543	SM	NP
16-2749	PG	NP
16-2931	JR	NP
16-3274	GS	NP
16-3321	SS	NP
16-3466	JD	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-3484	BLS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-3654	SK	MILD BALLONING DEGENERATION
16-3746	PS	NP
16-3753	VS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-3991	TS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-4146	JS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-4694	JS	UR
16-5056	SS	NP
16-5106	SK	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-5731	SS	BALLOONING DEGENERATION
16-5951	RK	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-5972	RB	NP
16-6290	ADG	NP
16-6352	GS	NP
16-6411	JS	NP
16-6473	AS	BALLOONING DEGENERATION
16-6575	PK	EMPERIPOLESIS AND HEPATOCYTIC ROSSETTING
16-6612	BK	MILD BALLONING DEGENERATION
16-6780	MK	MANY EPITHELIOD CELL GRANULOMAS WITH LANGHANS TYPE GIANT CELLS
16-7108	PR	NP
16-7744	SP	NP
16-7810	GS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-7858	JS	GROUND GLASS HEPATOCYTES
16-7943	SB	INFLAMMATORY GRANULATION WITH FIBROSTASIS PRILIFERATION. INFLAMMATORY CELLS ARE SEEN AS NEUTROPHILS, LYMPHOCYTES, PLASMA CELLS AND HISTIOCYTES.
16-7964	JK	FOCAL GROUND GLASS APPEARNACE
16-7999	PK	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-8330	HK	MILD BALLONING DEGENERATION
16-8313	KC	INTRAHEPATIC CHOLESTASIS WITH FEATHRY DEGENERATION MORE IN CENTRILOBULAR AREA
16-8476	NM	INTRAHEPATIC AND CANNALICULAR CHOESTASIS
16-8827	RK	NP
16-9096	G	GIANT CELL TRANSFORMATION NOTED
16-9234	CK	MILD BALLONING DEGENERATION

HISTO NO	INITIALS	INFLAMMATORY INFILTRATE PORTAL TRACTS/SEPTA/SINUSOIDS	PLASMA CELL RICH	GRANULOMA	INTERFACE HEPATITIS	SPOTTY HEPATIC NECROSIS
15-5619	IK	EXTRACELLULAR ACELLULAR MATERIAL	AB	AB	AB	AB
15-6236	DS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	P
15-6319	BS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	P	P
15-6463	RC	MILD CHRONIC INFLAMMATORY CELLS	AB	AB	P	P
15-7197	D	MILD MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, LYMPHOCYTES, FEW EOSINOPHILS AND OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
15-7269	RS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	P
15-7277	KS	MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES	AB	P	P	AB
15-7779	PS	MODERATE TO MARKED INFILTRATION BY LYMPHOCTRES, HISTOCYTES AND FEW EOSINOPHILS	AB	AB	P	P
15-7854	RP	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	AB
15-7903	NS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
15-7996	RK	MODERATE MONONUCLEAR CELL INFILTRATE	AB	AB	AB	AB
15-8123	KG	MODERATE MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, NEUTROPHILS, FEW PLASMA CELLS AND OCCASIONAL EOSINOPHILS	AB	AB	P	AB
15-8385	NS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS COMOSED OF LYMPOCYTES	AB	AB	AB	P
15-8387	KS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
15-8393	HS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	P
15-8703	MS	EDEMA WITH INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, NEUTROPHILS, HISTTOCYTES, EOSINOPHILS AND PLASMA CELLS	AB	AB	P	P
15-8959	GS	MODERATE INFILTRATION BY LYMPHOCYTES AND PLASMA CELLS	AB	AB	P	AB
15-9108	KK	MILD INFILTRATE SEEN AS LYMPHOCYTES, NEUTROPHILS, PLASMA CELLS AND HISTIOCYTES	AB	AB	P	AB
15-9150	SK	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	AB
15-9646	SK	MILD CHRONIC INFLAMMATORY CELLS	AB	AB	P	P
15-10166	GS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	P
15-10296	KR	MILD INFILTRATION SEEN AS LYMPHOCYTES, NEUTROPHILS AND HISTOCYTES	AB	AB	P	AB
15-11489	MS	MILD MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES AND PLASMA CELLS	AB	AB	AB	AB
15-11535	RM	MILD ACUTE AND CHRONIC INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES, PLASMA CELLS AND HISTOCYTES	AB	AB	AB	P
16-08	HK	DENSE INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES, EOSINOPHILS AND NEUTROPHILS	AB	AB	AB	AB
16-154	AG	MINIMAL INFILTRATION BY LYMPHOCYTES	AB	AB	AB	P
16-224	GS	MILD INFILTRATION BY LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
16-281	SK	INFILTRATION BY LYMPHOCYTES AND MONOCYTES	AB	AB	AB	AB
16-348	BS	MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, LYMPHOCYTES AND MACROPHAGES	AB	AB	P	AB
16-687	BS	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY CELLS COMPOSED OF LYMPHOCYTES	AB	AB	AB	P
16-828	NS	NO INFILTRATE	AB	AB	AB	P
16-1094	MK	MILD INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-1166	KS	MILD INFILTRATION BY LYMPHOCYTES	AB	AB	AB	P
16-1365	SK	ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
16-1487	KK	MILD CHRONIC INFLAMMATORY INFILTRATE WITH FOCAL INCREASE IN FIBROSIS	AB	AB	AB	P
16-1878	BS	MODERATE INFILTRATION BY ACUTE AND CHRONIC INFLAMMATORY CELLS	AB	AB	P	AB
16-2098	RS	DENSE LYMPHOMONONUCLEAR INFILTRATE	AB	AB	AB	AB
16-2191	RR	MILD LOBULAR INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-2355	JS	MILD CHRONIC INFLAMMATORY INFILTRATE BY LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
16-2413	SS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	AB
16-2543	SM	MODERATE DEGREE OF MIXED INFLAMMATORY INFILTRATE SEEN LYMPHOCYTES,FEW HISTIOCYTES, NEUTROPHILS AND OCCASIONAL EOSINOPHILS	AB	AB	P	P
16-2749	PG	MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES AND FEW NEUTROPHILS	AB	AB	P	AB
16-2931	JR	MODERATE DEGREE OF CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-3274	GS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	AB
16-3321	SS	NO INFILTRATE	AB	AB	AB	P
16-3466	JD	DENSE INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	P	AB
16-3484	BLS	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELLS COMPOSED OF LYMPHOCYTES MAINLY	AB	AB	AB	AB
16-3654	SK	NO INFILTRATE	AB	AB	AB	AB
16-3746	PS	MILD INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE WITH FOCAL INCREASE IN PORTAL FIBROSIS	AB	AB	AB	P
16-3753	VS	DENSE MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, NEUTROPHILS AND EOSINOPHILS	AB	AB	P	P
16-3991	TS	MILD INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	P
16-4146	JS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-4694	JS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-5056	SS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS COMPOSED OF LYMPHOCYTES MAINLY	AB	AB	AB	P
16-5106	SK	DENSE INFILTRATION BY LYMPHOMONONUCLEAR CELLS WITH PRESENCE OF PLASMA CELLS AND EOSINOPHILS.	AB	AB	P	P
16-5731	SS	MILD INFILTRATION BY LYMPHOMONONUCLER CELLS	AB	AB	AB	P
16-5951	RK	MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND NEUTROPHILS	AB	AB	P	AB
16-5972	RB	MILD NFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE	AB	AB	P	AB
16-6290	ADG	MINIMAL LYMPHOCYTIC INFILTRATION	AB	AB	AB	P
16-6352	GS	NO INFILTRATE	AB	AB	AB	P
16-6411	JS	MILD LYMPOVESCULAR CELLS INFILTRATE WITH OCCASIONAL LYMPHOID AGGREGATE	AB	AB	P	P
16-6473	AS	LYMPHOMONONUCLEAR CELL INFILTRATE	AB	AB	AB	P
16-6575	PK	DENSE INFILTRATION BY MILD INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW NEUTROPHILS AND PLASMA CELLS	AB	AB	P	AB
16-6612	BK	DENSE INFILTRATION BY PORTAL TRACTS BY MIXED INFLAMMATORY INFILTRATE COMPRISING LYMPHOCYTES, FEW NEUTROPHILS AND EOSINOPHILS.	AB	AB	AB	AB
16-6780	MK	INFILTRATED BY ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
16-7108	PR	MILD INFILTRATION BY CHRONIC LYMPHOMONONUCLEAR CELL INFILTRATE	AB	AB	AB	P
16-7744	SP	MILD INFILTRATION BY DENSE MIXED INFLAMMATORY MODERATE INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES AND FEW NEUTROPHILS, PLASMA CELLS AND OCCASIONAL EOSINOPHILS	AB	AB	P	P
16-7810	GS	DENSE MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, NEUTROPHILS, FEW HISTIOCYTES, EOSINOPHILS AND OCCASIONAL PLASMA CELLS	AB	AB	P	P
16-7858	JS	MILD LYMPHOCYTIC INFILTRATE	AB	AB	AB	P
16-7943	SB	NO INFILTRATE	AB	P	AB	AB
16-7964	JK	MILD INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-7999	PK	DENSE CHRONIC INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES AND PLASMA CELLS	AB	AB	P	P
16-8330	HK	MODERATE DEGREE OF INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES ADMIXED WITH FEW PLASMA CELLS.	AB	AB	FOCAL	AB
16-8313	KC	NO INFILTRATE	AB	AB	AB	AB
16-8476	NM	MILD CHRONIC INFILTRATE	AB	AB	AB	P
16-8827	RK	CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
16-9096	G	NO INFILTRATE	AB	AB	AB	AB
16-9234	CK	INFILTRATION BY CHRONIC INFLAMMATORY CELLS SEEN AS LYMPHOCYTES AND FEW HISTOCYTES	AB	AB	P	AB

HISTO NO	INITIALS	CONFLUENT NECROSIS	FIBROSIS	STAINS		
				IRON	MASSON TRICHOME	RETICULIN
15-5619	IK	AB	AB	ND	ND	ND
15-6236	DS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	ND
15-6319	BS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
15-6463	RC	AB	AB	NEGATIVE	HIGHLIGHTS FIBROSIS IN OCCASIONAL PORTAL TRACT	MAINTAINED
15-7197	D	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-7269	RS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-7277	KS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-7779	PS	AB	AB	NEGATIVE	HIGHLIGHTS FIBROSIS AND NODULE FORMATION	MAINTAINED
15-7854	RP	AB	AB	NEGATIVE	ND	ND
15-7903	NS	AB	AB	NEGATIVE	HIGHLIGHTS INCREASE IN PORTAL AND PERICELLULAR FIBROSIS	MAINTAINED
15-7996	RK	AB	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS AND BROAD SCAR	MAINTAINED
15-8123	KG	AB	GRADE II	NEGATIVE	HIGHLIGHTS PORTO PORTAL SEPTAL	MAINTAINED
15-8385	NS	AB	GRADEII	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-8387	KS	AB	GRADE III	NEGATIVE	HIGHLIGHTS PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
15-8393	HS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-8703	MS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-8959	GS	AB	GRADE III	NEGATIVE	HIGHLIGHTS PERICELLULAR AND PORTAL FIBROSIS	COLLAPSE
15-9108	KK	AB	GRADE II	NEGATIVE	PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
15-9150	SK	AB	ABSENT	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-9646	SK	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
15-10166	GS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
15-10296	KR	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
15-11489	MS	AB	GRADE II	NEGATIVE	PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
15-11535	RM	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
16-08	HK	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-154	AG	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-224	GS	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS	MAINTAINED
16-281	SK	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS	MAINTAINED
16-348	BS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
16-687	BS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-828	NS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-1094	MK	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-1166	KS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS	FOCAL COLLAPSE
16-1365	SK	AB	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS	MAINTAINED
16-1487	KK	AB	GRADE II	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
16-1878	BS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
16-2098	RS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS FIBROSIS	MAINTAINED
16-2191	RR	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL AND PERICELLULAR FIBROSIS	MAINTAINED
16-2355	JS	AB	GRADE I	NEGATIVE	HIGHLIGHTS ORTAL FIBROSIS AND PERIPORTAL FIBROSIS	MAINTAINED
16-2413	SS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
16-2543	SM	AB	GRADE II	NEGATIVE	HIGHLIGHTS WITH FIBROUS SEPTAL	MAINTAINED
16-2749	PG	AB	GRADE IV	NEGATIVE	HIGHLIGHTS FIBROSIS AND NODULE FORMATION	MAINTAINED
16-2931	JR	AB	GRADE I	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
16-3274	GS	AB	GRADE II	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	MAINTAINED
16-3321	SS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-3466	JD	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS	MAINTAINED
16-3484	BLS	AB	GRADE II	NEGATIVE	HIGHLIGHTS FIBROSIS AND NODULE FORMATION	MAINTAINED
16-3654	SK	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERICELLULAR AND PERIVASCULAR FIBROSIS	MAINTAINED
16-3746	PS	AB	GRADE I	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
16-3753	VS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS AND NODULE FORMATION	MAINTAINED
16-3991	TS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL AND PERIPORTAL FIBROSIS	MAINTAINED
16-4146	JS	AB	GRADE II	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS IN IN OCCASIONAL PORTAL TRACT	MAINTAINED
16-4694	JS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-5056	SS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
16-5106	SK	AB	GRADE IV	FOCAL	HIGHLIGHTS NODULE FORMATION AND INCREASED	MAINTAINED
16-5731	SS	AB	AB	NEGATIVE	DOES NOT SHOW INCREASED FIBROSIS	MAINTAINED
16-5951	RK	AB	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
16-5972	RB	AB	GRADE II	NEGATIVE	HIGHLIGHTS PORTO PORTAL FIBROSIS	MAINTAINED
16-6290	ADG	AB	GRADE II	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
16-6352	GS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS INCREASED FIBROSIS	MAINTAINED
16-6411	JS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
16-6473	AS	AB	GRADE II	NEGATIVE	HIGHLIGHTS FOCAL PERIVENULAR FIBROSIS	MAINTAINED
16-6575	PK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS AND ATTEMPTED NODULE FORMATION	MAINTAINED
16-6612	BK	AB	GRADE II	NEGATIVE	HIGHLIGHTS FOCAL PERICELLULAR FIBROSIS	MAINTAINED
16-6780	MK	AB	AB	ND	ND	ND
16-7108	PR	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTO PORTAL FIBROSIS	MAINTAINED
16-7744	SP	P	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS THE PORTO PORTAL SEPTA	WITH PERICELLULAR FIBROSIS
16-7810	GS	P	GRADE III	NEGATIVE	INCREASED PORTAL FIBROSIS WITH PORTO PORTAL SEPTAL	MAINTAINED
16-7858	JS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-7943	SB	AB	AB	NEGATIVE	ND	ND
16-7964	JK	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
16-7999	PK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND FIBROSIS	MAINTAINED
16-8330	HK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION WITH FOCAL PERICELLULAR FIBROSIS	MAINTAINED
16-8313	KC	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS FIBROSIS	MAINTAINED
16-8476	NM	AB	AB	NEGATIVE	DO NOT HIGHLIGHT INCREASED PORTAL FIBROSIS	MAINTAINED
16-8827	RK	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
16-9096	G	AB	AB	NOT DONE	NOT DONE	NOT DONE
16-9234	CK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND PERICELLULAR FIBROSIS	MAINTAINED

HISTO NO	INITIALS	OTHERS
15-5619	IK	CONGO RED STAIN FOR AMYLOID IS POSITIVE
15-6236	DS	ND
15-6319	BS	ND
15-6463	RC	ND
15-7197	D	ND
15-7269	RS	ND
15-7277	KS	ZN & AFB STAIN IS NEGATIVE
15-7779	PS	ND
15-7854	RP	ND
15-7903	NS	ND
15-7996	RK	ND
15-8123	KG	ORCEIN AND RHODENINE STAIN SHOW ACCUMULATION OF COPPER GRANULAR IN HEPATOCYTES
15-8385	NS	ND
15-8387	KS	ND
15-8393	HS	ND
15-8703	MS	ND
15-8959	GS	ND
15-9108	KK	ND
15-9150	SK	ORCEIN AND RHODENINE STAIN NEGATIVE
15-9646	SK	AB
15-10166	GS	AB
15-10296	KR	AB
15-11489	MS	ND
15-11535	RM	ND
16-08	HK	ND
16-154	AG	ND
16-224	GS	ND
16-281	SK	ND
16-348	BS	ND
16-687	BS	ND
16-828	NS	ND
16-1094	MK	ND
16-1166	KS	ND
16-1365	SK	ND
16-1487	KK	ND
16-1878	BS	ND
16-2098	RS	ND
16-2191	RR	ND
16-2355	JS	ND
16-2413	SS	ND
16-2543	SM	ND
16-2749	PG	ND
16-2931	JR	ND
16-3274	GS	ND
16-3321	SS	ND
16-3466	JD	ND
16-3484	BLS	ORECIN AND RHODODENDRON FOR COPPER SHOW MILD FOCAL PRESENCE OF COPPER IN PERISEPTAL REGION
16-3654	SK	ND
16-3746	PS	ND
16-3753	VS	ND
16-3991	TS	ND
16-4146	JS	ND
16-4694	JS	ND
16-5056	SS	ND
16-5106	SK	ND
16-5731	SS	ND
16-5951	RK	ND
16-5972	RB	ND
16-6290	ADG	ND
16-6352	GS	ND
16-6411	JS	ORECIN FOR COPPER IS NEGATIVE
16-6473	AS	ND
16-6575	PK	ND
16-6612	BK	ND
16-6780	MK	ZN STAIN FOR AFB IS NEGATIVE
16-7108	PR	ND
16-7744	SP	ND
16-7810	GS	ND
16-7858	JS	ND
16-7943	SB	ZN STAIN FOR AFB IS NEGATIVE
16-7964	JK	ND
16-7999	PK	ND
16-8330	HK	ND
16-8313	KC	ND
16-8476	NM	ND
16-8827	RK	ND
16-9096	G	ND
16-9234	CK	ND

HISTO NO	INITIALS	IMPRESSION
15-5619	IK	AMYLOIDOSIS
15-6236	DS	NASH GRADE 2 STAGE 2
15-6319	BS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 1 GRADE 1, STAGE 0
15-6463	RC	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4, GRADE 1 STAGE 1
15-7197	D	PRIMARY BILIARY CIRRHOSIS
15-7269	RS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 3 GRADE 1, STAGE 0
15-7277	KS	TUBERCULOSIS
15-7779	PS	PRIMARY BILIARY CIRRHOSIS
15-7854	RP	AMYLOIDOSIS
15-7903	NS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) HAI SCORE 2, GRADE 1,STAGE 1
15-7996	RK	ASH GRADE3 STAGE 4
15-8123	KG	PRIMARY BILIARY CIRRHOSIS
15-8385	NS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 2 GRADE 1, STAGE 1
15-8387	KS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 3 GRADE 1, STAGE 0
15-8393	HS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 3, GRADE 1 STAGE 1
15-8703	MS	CHOLESTATIC LIVER DISEASE
15-8959	GS	ASH GRADE6 STAGE 3
15-9108	KK	NASH GRADE 3 STAGE 2
15-9150	SK	METABOLIC LIVER DISEASE – DUBIN JONHSON SYNDROME
15-9646	SK	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4, GRADE 2 STAGE 1
15-10166	GS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 1, GRADE 1 STAGE 1
15-10296	KR	NASH GRADE 3 STAGE 2
15-11489	MS	NASH GRADE 6 STAGE 1
15-11535	RM	ASH GRADE 5 STAGE 3
16-08	HK	LARGE DUCT OBSTRUCTION
16-154	AG	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1, STAGE II
16-224	GS	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) HAI SCORE 4,GRADE 2,STAGE 4
16-281	SK	CIRRHOSIS– IDIOPATHIC
16-348	BS	NO SPECIFIC PATHOLOGY
16-687	BS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 3, GRADE 1 STAGE 0
16-828	NS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 1, GRADE ,1 STAGE 0
16-1094	MK	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 1, GRADE 1 STAGE 0.
16-1166	KS	ACUTE HEPATITIS (HEV POSITIVE)
16-1365	SK	AUTOIMMUNE HEPATITIS
16-1487	KK	CHRONIC VIRAL HEPATITIS(HCV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 2, STAGE 1
16-1878	BS	CIRRHOSIS – IDIOPATHIC
16-2098	RS	LARGE DUCT OBSTRUCTION
16-2191	RR	NASH GRADE 3 STAGE 1
16-2355	JS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4, GRADE 2 STAGE 2
16-2413	SS	CHRONIC VIRAL HEPATITIS GOING ON TO CIRRHOSIS (HBV POSITIVE) HAI SCORE 2 GRADE 1 STAGE 3
16-2543	SM	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MODERATE ACTIVITY HAI SCORE 8, GRADE 2 STAGE 3
16-2749	PG	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4,GRADE 2, STAGE 1
16-2931	JR	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) HAI SCORE 3, GRADE 1,STAGE 1
16-3274	GS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 1, GRADE 1, STAGE 1
16-3321	SS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1, STAGE 0
16-3466	JD	AUTOIMMUNE HEPATITIS
16-3484	BLS	LARGE DUCT OBSTRUCTION
16-3654	SK	NASH GRADE 5 STAGE 2
16-3746	PS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 3, GRADE 1, STAGE 2
16-3753	VS	DILI (ATT)
16-3991	TS	CHRONIC HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2 GRADE 1, STAGE 2
16-4146	JS	CHRONIC HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2 GRADE 1, STAGE 1
16-4694	JS	NO SPECIFIC PATHOLOGY
16-5056	SS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 5 GRADE 2 STAGE 1
16-5106	SK	CIRRHOSIS WITH ACTIVITY- IDIOPATHIC
16-5731	SS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 1, GRADE 1, STAGE 1
16-5951	RK	PRIMARY SCLEROSING CHOLANGITIS
16-5972	RB	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4, GRADE 2, STAGE 2
16-6290	ADG	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 2 GRADE 1, STAGE 1
16-6352	GS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 1 GRADE 1, STAGE 0
16-6411	JS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 3 GRADE 1, STAGE 2
16-6473	AS	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1, STAGE II
16-6575	PK	AUTOIMMUNE HEPATITIS
16-6612	BK	NASH GRADE 5 STAGE 1
16-6780	MK	GRANULOMATOUS INFLAMMATION - TUBERCULOSIS
16-7108	PR	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 2 GRADE 1 STAGE 2
16-7744	SP	CHRONIC VIRAL HEPATITIS – AUTOIMMUNE WITH PBC LIKE FEATURES- OVERLAP SYNDROME
16-7810	GS	AUTOIMMUNE HEPATITIS
16-7858	JS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1 STAGE 0
16-7943	SB	GRANULOMATOUS INFLAMMATION -TUBERCULOSIS
16-7964	JK	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY(HAI SCORE 3,GRADE 1,STAGE0
16-7999	PK	AUTOIMMUNE HEPATITIS
16-8330	HK	NASH GRADE 3 STAGE 4
16-8313	KC	ACUTE HEPATITS(HEV)
16-8476	NM	ACUTE HEPATITIS(HAV POSITIVE)
16-8827	RK	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2 GRADE 1, STAGE 0
16-9096	G	CHOLESTATIC LIVER DISEASE- SEPSIS INDUCED
16-9234	CK	NASH GRADE 2 STAGE 4

				HISTORY									BIOCHEMISTRY								
HISTO NO	INITIALS	MRD	AGE/SEX	JAUNDICE	DIABETES	ALCOHAL	OBESITY	ABDOMINAL MASS	HEPATO MEGALY	SPLENOME GALY	LYMPHADENO PATHY	RADIOLOGICAL FINDINGS	BILI TOTAL	BILI DIRECT	SGOT	SGPT	ALBUMIN	ALP	PROTIENS	VIRAL MARKERS	AUTOIMMUNE MARKERS
16-9315	SD	133512	69/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	35	45	3.5	120	6.8	NR	NEGATIVE
16-9793	M	131346	11/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	40	45	3.2	110	6.5	NR	ND
16-10131	GS	146530	14/M	P	AB	AB	AB	AB	P	P	AB	ND	12.5	10.2	50	60	5	200	8	NR	ND
16-10668	DS	39640	34/M	AB	AB	AB	AB	AB	P	AB	AB	ND	4.5	4	40	45	4	110	6.5	NR	ANCA 1+
16-11131	AA	128428	25/M	AB	AB	AB	AB	AB	AB	AB	nd	ND	1	0.3	35	45	3.6	115	6.4	HBV POSITIVE	NEGATIVE
16-11127	GS	145278	17/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	30	40	3	112	6	HCV-POSITIVE	NEGATIVE
16-11701	GS	136357	25/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	8.8	7.5	60	72	4.5	210	5	NR	ANCA 1+
16-11965	RL	139332	65/M	AB	AB	AB	AB	AB	AB	P	AB	HEPATOMEGALY	2	0.4	70	55	4	100	6	NR	NEGATIVE
16-12253	VKA	33490	64/M	AB	AB	AB	P	AB	P	AB	AB	HEPATOMEGALY	1.5	1.2	55	45	4	102	5.6	NR	NEGATIVE
16-12407	RD	113510	29/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	12.1	10.2	40	50	4.5	90	6.8	HBV-POSITIVE	NEGATIVE
16-12459	JR	149158	46/M	P	AB	AB	AB	AB	AB	AB	AB	ND	10.2	8.5	70	75	5.2	210	5.2	NR	ND
16-12560	GS	247500	26/M	AB	AB	AB	P	AB	P	AB	AB	HEPTOMEGALY	1.5	0.9	50	55	5.6	150	8	NR	ND
16-12508	DR	95201	60/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.2	0.5	40	45	4.5	120	6.5	NR	ND
'16-12826	AR	153198	58/F	P	AB	AB	AB	AB	P	AB	AB	CHOLESTATIC LIVER DISEASE	13.6	12.5	70	80	5	210	6.8	NR	NEGATIVE
16-13009	RK	49468	37/M	AB	AB	AB	AB	AB	P	P	AB	ND	15.6	12.2	76	80	5	126	7.9	NR	ANA 2+, AMA 2+, SNA 1+, ASMA 3+ LKM 1+
16-13132	BK	159521	75/F	P	AB	AB	AB	AB	P	AB	AB	ND	30.6	25.5	110	126	5	310	6.9	NR	ND
16-13091	RA	100986	45/F	AB	P	AB	AB	AB	AB	AB	AB	ND	6	4.9	50	55	4.2	210	7.8	NR	ANA3+, SMA 3+, AMA 3+,LKM2+
17-03	RK	170653	61/M	P	AB	AB	AB	AB	AB	AB	AB	ND	4.25	3.59	35	46	5.4	600	3.7	NR	ND
17-196	SS	166885	38/M	P	AB	AB	AB	AB	P	AB	AB	ND	27.1	13.9	258	142	3.06	195	7.5	NR	NEGATIVE
17-425	RS	180277	53/M	P	AB	AB	AB	AB	AB	AB	AB	ND	31.9	20.9	75	26	2.4	147	8.2	NR	ND
17-471	BK	178668	55/F	P	P	AB	P	AB	AB	AB	AB	ND	26.19	12.32	99	62	3.05	128	3.5	NR	ND
17-534	PD	181547	23/F	AB	AB	AB	AB	AB	P	P	AB	HEPATOMEGALY	5.7	4.86	319	133	2.3	127	8.1	NR	NEGATIVE
17-775	NG	185669	52/F	P	AB	AB	AB	AB	AB	AB	AB	ND	10.28	5.5	62	25	4.5	343	7.5	NR	NEGATIVE
17-1062	RK	186406	40/M	P	AB	P	AB	AB	P	AB	AB	HEPATOMEGALY	15.2	12.9	130	39	2.6	152	7.3	NR	NEGATIVE
17-1185	GC	189051	60/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.42	0.33	52	15	3	552	7	NR	ND
17-1248	RSA	194154	56/M	P	P	P	AB	AB	AB	AB	AB	FATTY LIVER	40	20.29	107	37	3.15	230	5.6	NR	ND
17-1326	DS	191742	46/M	P	AB	P	AB	AB	AB	AB	AB	FATTY LIVER	39	31	153	70	9	315	7.5	NR	NEGATIVE
17-1382	KS	190310	49/M	AB	AB	P	P	AB	P	AB	AB	FATTY LIVER	0.69	0.27	158	247	4.4	757	7.2	NR	ANA 1+
17-1417	PS	191741	24/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.5	1.2	29	28	3.5	130	8	HBC-POSITIVE	ND
17-1470	RK	199232	55/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.7	0.17	34	20	3.36	371	9.5	NR	ND
17-1623	NM	201073	34/F	P	AB	AB	AB	AB	AB	AB	AB	ND	4.8	2.56	145	132	3.58	1103	7.9	NR	ND
17-1700	VG	153505	39/M	AB	AB	P	P	AB	P	P	AB	ND	5.07	3.26	67	26	2.3	144	6.8	NR	ND
17-1856	M	206642	47/F	AB	AB	AB	P	AB	P	AB	AB	ND	2	0.5	50	55	6	150	7	NR	ANA 1+
17-1966	PD	205027	20/F	P	AB	AB	AB	AB	AB	AB	AB	ND	8	10	120	150	5	200	6	HAV-POSITIVE	NEGATIVE
17-2239	BS	210994	30/M	AB	AB	AB	P	AB	P	P	AB	HEPATOSPLEENOMEGALY	2	1	60	65	3.5	180	6.8	NR	NEGATIVE
17-2397	HS	191338	57/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	3	2	60	70	4	102	7	NR	ANA3+, AMA2+, ANCA 2+
17-2508	AK	214427	73/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	60	65	3.5	100	7	NR	ND
17-2617	KL	166916	44/M	AB	AB	AB	AB	AB	P	P	AB	ND	1	0.2	50	55	4	110	6	HBV-POSITIVE	ND
17-2615	IG	209332	17/F	P	AB	AB	AB	AB	AB	AB	AB	ND	15	18	60	70	5	280	6	HAV-POSITIVE	ANA 1+, ASMA 1+, ANCA 1+
17-2683	AS	219598	28/M	AB	AB	P	AB	AB	P	AB	AB	ND	1	0.2	40	45	5	60	7	NR	ND
17-2981	DD	183018	48/F	P	AB	AB	AB	AB	AB	AB	AB	ND	1.54	1.07	128	121	4.1	74	8.3	HBV POSITIVE	ND
17-3005	SPK	415425	69/F	P	P	AB	AB	AB	AB	AB	AB	ND	0.21	0.08	14	9	3.4	62	6.7	NR	ANA 2+ AMA 2+ ASMA 1+ LKM 1+
17-3041	AJ	222747	62/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	2	2.5	140	100	4	180	6.5	NR	ND
17-3132	MS	168096	45/ M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5	3	60	70	4	140	8	NR	AMA 1+, ANCA 3+
17-3336	A	175262	39/M	P	AB	AB	AB	AB	AB	AB	AB	ND	6	5.5	100	110	5	140	8	NR	ND
17-3456	HK	237267	43/F	AB	AB	AB	P	AB	AB	AB	AB	ND	4	4.5	70	75	6	150	9	NR	ANA 3+, ANCA 3+, LKM 2+
17-4152	SR	58867	57/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.5	5.2	120	150	4.2	162	7.5	NR	ND
17-4567	SP	251541	60/M	AB	AB	P	AB	AB	AB	AB	AB	ND	3.5	3.2	140	146	5.6	200	6.7	NR	ND
17-4676	ML	252417	56/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.6	5	182	190	3.5	152	6.2	NR	ND
17-4738	N	257384	23/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.5	1.2	82	85	2.5	120	5.6	NR	ND
17-4995	J	260987	36/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.2	1	150	158	2	240	5	NR	ND
17-5095	RK	254704	52/M	AB	AB	P	AB	AB	AB	AB	AB	ND	10.5	10.2	90	100	4.5	152	4.5	NR	ND
17-5253	DR	204939	58/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	2.2	1.9	100	110	2.5	250	6.5	HCV POSITIVE	ND
17-5497	KK	264927	57/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	4.2	3.8	126	150	2.6	300	5.5	NR	ND
17-5723	VK	146862	56/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	3.8	3.5	150	136	2.1	150	5	NR	ND
17-5928	RK	151220	44/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	2.5	2.1	148	142	2	142	4.2	NR	ND
17-6520	B	36925	59/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	2	1.8	80	85	1.8	0	3.8	NR	ND
'17-6825	SK	281066	62/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.8	1.2	125	120	1.6	224	4.2	NR	ND
17-6962	AK	289068	17/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.2	1	142	156	1	152	2.6	NR	ND
17-7018	AS	267338	19/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.5	1.2	152	140	1.5	180	2	NR	ND
17-7338	MK	287091	60/F	AB	AB	AB	AB	AB	AB	AB	AB	GRANULOMATOUS INFLAMMATION	2.2	2	250	275	2.5	250	2.1	NR	ND
17-8000	BS	304749	48/M	P	AB	AB	AB	AB	AB	AB	AB	ND	5.6	5.2	180	195	3.6	110	6	NR	ND
17-8496	SK	313774	49/F	AB	P	AB	AB	AB	AB	AB	AB	ND	6.2	6	200	210	4	300	4.6	NR	ND
17-8495	JS	313629	32/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	8.5	8.2	250	230	3.2	258	4	NR	ND
17-8664	HS	307336	24/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.2	5	160	182	2.2	112	3.5	HBV POSITIVE	ND
17-8732	SK	311346	43/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISAESE	8.2	8	220	234	2	200	3.2	NR	ND
17-9013	MGP	320257	56/M	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	2.5	2.2	150	150	1.8	143	3.6	NR	ND
17-9239	HT	323120	58/M	AB	AB	AB	AB	P	AB	AB	AB	ND	3	3.5	180	160	3.2	200	3	NR	ND
17-9361	HS	328005	15/M	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	2.5	2.8	120	132	4.5	160	2.5	NR	ND
17-9710	JS	332720	37/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.2	1	158	162	2.3	186	4	NR	ND
17-9820	V	295609	12/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.8	0.6	250	265	4	200	5.2	NR	ND
17-10174	RS	54829	46/M	P	AB	AB	AB	AB	AB	AB	AB	ND	2	1.5	35	40	4	130	7	NR	AMA 3+
17-10377	SP	64634	41/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.2	25	30	3.5	100	7	NR	ND
17-10474	UR	82060	66/F	P	AB	AB	AB	AB	P	AB	AB	HEPATOSPLEENOMEGALY	0.8	0.6	35	45	4	80	7	NR	ND
17-10502	SKG	291856	58/M	AB	P	AB	AB	AB	AB	AB	AB	ND	0.6	0.4	40	45	5	120	8	NR	ND
17-10504	HKK	342390	60/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.3	35	40	4	100	6	NR	ND
17-10537	SS	343852	33/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.72	0.44	40	72	4.3	72	8.1	NR	ND
17-1936	ND	196313	56/F	P	AB	AB	AB	AB	AB	AB	AB	ND	1.8	1.1	50	21	3.1	153	7	NR	ND

HISTO NO	INITIALS	CERULOPLASMIN	IRON	LIVER BIOPSY GROSS NUMBER	LENGTH	ARCHITECTURE	FATTY CHANGE TYPE	FATTY CHANGE DEGREE
16-9315	SD	ND	ND	2	1 & 1.5 CM	MILD LOBULAR DISARRY	MACROVESICULAR	0.4
16-9793	M	ND	ND	1	1.2 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	0.4
16-10131	GS	112	100	1	0.4 CM	DISARRAY	AB	AB
16-10668	DS	28	120	1	1.2 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-11131	AA	ND	ND	1	1.5 CM	MAINTAINED	AB	AB
16-11127	GS	ND	ND	2	1.2, 1.5 CM	MAINTAINED	AB	AB
16-11701	GS	ND	ND	2	0.3 & 0.4 CM	LOBULAR DISARRAY	AB	AB
16-11965	RL	ND	ND	3	1, 0.8, 0.2 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.1
16-12253	VKA	ND	ND	1	0.4 CM	MILD DISARRAY	MACROVESICULAR	0.4
16-12407	RD	ND	ND	1	0.7 CM	MAINTAINED	AB	AB
16-12459	JR	26	50	1	1 CM	MAINTAINED	AB	AB
16-12560	GS	ND	ND	1	2.4 CM	MAINTAINED	MACROVESICULAR	0.25
16-12508	DR	ND	ND	1	1.5 CM	MAINTAINED	MACROVESICULAR	0.75
'16-12826	AR	ND	ND	6	0.2 TO 0.4 CM	DISTORTED ARCHITECTURE WITH PRESENCE OF BROAD SCAR	AB	AB
16-13009	RK	28	ND	1	1.3 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
16-13132	BK	ND	ND	1	1.2 CM	MAINTAINED	MACROVESICULAR	0.9
16-13091	RA	28	65	1	1 CM	MAINTAINED	AB	AB
17-03	RK	ND	ND	1	1.3 CM	MAINTAINED	AB	AB
17-196	SS	30	101	2	0.2 & 0.4 CM	DISTORTED	AB	AB
17-425	RS	ND	ND	4	0.1 & 0.2 CM	DISARRAY WITH PRESENCE OF BROAD PORTAL SCAR	MACROVESICULAR	0.25
17-471	BK	ND	ND	1	0.8 CM	DISTORTED WITH PORTAL SCAR AND NODULE FORMATION	AB	AB
17-534	PD	ND	ND	2	0.2, 0.6 CM	DISTORTED WITH LOBULAR DISARRAY	AB	AB
17-775	NG	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	AB	P
17-1062	RK	ND	ND	2	1 & 1.2 CM	DISTORTED WITH NODULE FORMATION AND THICK SCAR FORMATION	MACROVESICULAR	0.1
17-1185	GC	ND	ND	1	2 CM	MAINTAINED	MACROVESICULAR	0.2
17-1248	RSA	ND	ND	1	0.8 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.7
17-1326	DS	ND	ND	1	0.6 CM	DISTORTED ARCHITECTURE WITH PRESENCE OF PORTO PORTAL FIBROSIS	MACROVESICULAR	0.75
17-1382	KS	28	ND	1	0.8 CM	MAINTAINED	MACROVESICULAR	0.25
17-1417	PS	ND	ND	1	1.2 CM	MAINTAINED	MACROVESICULAR	0.1
17-1470	RK	ND	ND	2	0.2 & 1 CM	MAINTAINED	AB	AB
17-1623	NM	ND	ND	1	1.2 CM	DISARRAY OF PORTAL ARCHITECTURE WITH ATTEMPTED NODULE FORMATION AND INCREASED PORTAL FIBROSIS	AB	AB
17-1700	VG	20	ND	1	2 CM	DISTORTED WITH NODULAR FORMATION	MACROVESICULAR	0.7
17-1856	M	ND	ND	1	1.5 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.8
17-1966	PD	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
17-2239	BS	20	ND	2	0.1, 0.1 CM	DISTORTED	MACROVESICULAR	0.3
17-2397	HS	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
17-2508	AK	ND	ND	2	0.2 & 0.2 CM	MAINTAINED	AB	AB
17-2617	KL	ND	ND	2	0.4 CM& 0.5 CM	MAINTAINED	AB	AB
17-2615	IG	ND	ND	1	1.3 CM	LOBULAR DISARRAY	AB	AB
17-2683	AS	ND	ND	4	0.2 TO 0.3 CM	DISTORTED WITH NODULAR FORMATION BY THICK FIBROUS SEPTA	MACROVESICULAR	0.7
17-2981	DD	ND	ND	1	1.5 CM	DISTORTED WITH NODULE FORMATION	AB	AB
17-3005	SPK	ND	ND	1	1.7 CM	DISTORTED WITH OCCASIONAL ATTEMPTED NODULES FORMATION	AB	AB
17-3041	AJ	35	ND	1	1.2 CM	DISTORTED WITH PRESENCE OF BRIDGING FIBROSIS	MACROVESICULAR	0.5
17-3132	MS	25	ND	1	1.5 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICKENED FIBROUS SEPTAL	MACROVESICULAR	0.05
17-3336	A	ND	ND	1	1.6 CM	MAINTAINED	MACROVESICULAR	0.5
17-3456	HK	ND	ND	1	1.2 CM	LOBULAR DISARRAY	AB	AB
17-4152	SR	ND	ND	1	1.4CM	DISTORTED WITH NODULE FORMATION	AB	AB
17-4567	SP	ND	ND	1	2 CM	DISTORTED WITH BROAD PORTAL SCAR	MACROVESICULAR	0.7
17-4676	ML	ND	ND	1	2.5 CM	DISTORTED WITH OCCASIONAL ATTEMPTED NODULE FORMATION	AB	AB
17-4738	N	ND	ND	1	2.5 CM	MAINTAINED	AB	AB
17-4995	J	ND	ND	4	0.1 TO 0.2 CM	DISTORTED WITH NODULE FORMATION ALONG WITH PRESENCE OF BROAD PORTAL SCAR	AB	AB
17-5095	RK	ND	ND	3	0.1,0.2 & 0.3 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
17-5253	DR	ND	ND	1	0.7 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION THICK AND THIN FIBROUS SEPTAL	AB	AB
17-5497	KK	ND	ND	1	0.8 CM	DISTORTED WITH PRESENCE OF BROAD PORTAL SCAR	MACROVESICULAR	AB
17-5723	VK	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
17-5928	RK	ND	ND	2	0.1 & 0.1 CM	DISTORTED WITH PORTAL FIBROSIS AND THICK PORTO PORTAL SEPTAL	AB	AB
17-6520	B	ND	ND	1	1 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.1
'17-6825	SK	ND	ND	1	1.2 CM	ARCHITECTURAL DISTORTION	AB	AB
17-6962	AK	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION AND BROAD SCAR	AB	AB
17-7018	AS	ND	ND	1	1.2 CM	DISARRAY WITH OCCASIONAL ATTEMPTED NODULE FORMATION	AB	AB
17-7338	MK	ND	ND	1	1CM	MILD DISARRAY OF LOBULAR ARCHITECTURE	AB	AB
17-8000	BS	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.35
17-8496	SK	ND	ND	2	0.1 & 0.2 CM	DISTORTED WITH NODULE FORMATION	AB	AB
17-8495	JS	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
17-8664	HS	ND	ND	1	1 CM	MAINTAINED WITH PORTAL PORTAL SEPTAL	AB	AB
17-8732	SK	ND	ND	3	0.2, 0.3 & 0.1 CM	DISTORTED WITH NODULE FORMATION AND PORTAL SCARY	AB	AB
17-9013	MGP	ND	ND	1	1.5 CM	DISTORTED WITH PORTAL FIBROSIS AND SCARS AND NODULE FORMATION	MACROVESICULAR	0.1
17-9239	HT	ND	ND	2	0.1 & 0.2 CM	MAINTAINED	AB	AB
17-9361	HS	ND	ND	1	1.2 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION AND PORTAL SCARS	AB	AB
17-9710	JS	ND	ND	2	0.3 & 0.5 CM	MAINTAINED	MACROVESICULAR	0.25
17-9820	V	ND	ND	1	1.2 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.5
17-10174	RS	ND	ND	2	1.2 & 1.5 CM	MAINTAINED ARCHITECTURE	AB	AB
17-10377	SP	ND	ND	1	1 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
17-10474	UR	ND	ND	1	0.7 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICKENED FIBROUS SEPTAL	MACROVESICULAR	0.4
17-10502	SKG	ND	ND	1	1.2CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.3
17-10504	HKK	ND	ND	2	1.2 & 1.5 CM	LOBULAR DISARRAY WITH ARCHITECTURE DISTORTION	MACROVESICULAR	0.2
17-10537	SS	ND	ND	1	0.1 TO 0.2 CM	ARCHITECTURAL DISTORTION WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.05
17-1936	ND	ND	ND	2	0.2 TO 0.5 CM	NOT MENTIONED IN REPORT	AB	AB

HISTO NO	INITIALS	CHOLESTASIS	BILE	BILE DUCT	MALLORY DENK BODIES
16-9315	SD	AB	P	AB	AB
16-9793	M	AB	AB	AB	AB
16-10131	GS	AB	AB	AB	AB
16-10668	DS	P	AB	AB	AB
16-11131	AA	AB	AB	AB	AB
16-11127	GS	AB	AB	AB	AB
16-11701	GS	CANALICULAR	BILE PLUGS	P	AB
16-11965	RL	AB	AB	REACTION	P
16-12253	VKA	AB	AB	AB	P
16-12407	RD	AB	AB	AB	AB
16-12459	JR	INTRA CELLULAR	BILEPLUGS	P	AB
16-12560	GS	AB	AB	AB	AB
16-12508	DR	AB	AB	AB	AB
'16-12826	AR	AB	BILE DUCT PRILIFERATION	AB	AB
16-13009	RK	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
16-13132	BK	INTRA CELLULAR AND CAVALICULAR TYPE	AB	AB	AB
16-13091	RA	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-03	RK	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-196	SS	AB	STASIS	AB	AB
17-425	RS	AB	STASIS	AB	P
17-471	BK	P	AB	AB	AB
17-534	PD	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-775	NG	AB	AB	PROLIFERATION	AB
17-1062	RK	AB	AB	AB	AB
17-1185	GC	AB	AB	AB	AB
17-1248	RSA	AB	P	AB	P
17-1326	DS	INTRAHEPATIC	BILE PLUGS	AB	P
17-1382	KS	INTRACELLULAR	STASIS	AB	AB
17-1417	PS	AB	AB	AB	AB
17-1470	RK	AB	AB	AB	AB
17-1623	NM	AB	AB	AB	AB
17-1700	VG	AB	AB	AB	P
17-1856	M	AB	BILE PLUGS	AB	AB
17-1966	PD	AB	BILE PLUGS	AB	AB
17-2239	BS	AB	AB	AB	AB
17-2397	HS	AB	AB	AB	AB
17-2508	AK	AB	INTRACELLULAR	AB	AB
17-2617	KL	AB	AB	AB	AB
17-2615	IG	AB	INTRACELLULAR	P	AB
17-2683	AS	AB	BILE PLUGS	DUCTULAR PROLIFERATION NOTED	AB
17-2981	DD	AB	AB	DUCTULAR PROLIFERATION	AB
17-3005	SPK	AB	AB	AB	AB
17-3041	AJ	AB	AB	AB	AB
17-3132	MS	AB	AB	AB	AB
17-3336	A	AB	AB	AB	AB
17-3456	HK	AB	AB	AB	AB
17-4152	SR	AB	AB	AB	AB
17-4567	SP	AB	AB	DUCTULAR PROLIFERATION NOTED	P
17-4676	ML	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-4738	N	AB	AB	AB	AB
17-4995	J	AB	AB	DUCTULAR PROLIFERATION NOTED	P
17-5095	RK	AB	P	PROMINENT	AB
17-5253	DR	AB	AB	AB	AB
17-5497	KK	AB	AB	AB	P
17-5723	VK	AB	INTRACELLULAR	AB	AB
17-5928	RK	AB	AB	AB	AB
17-6520	B	AB	AB	AB	AB
'17-6825	SK	AB	AB	AB	P
17-6962	AK	AB	AB	AB	AB
17-7018	AS	AB	AB	AB	AB
17-7338	MK	AB	AB	AB	AB
17-8000	BS	AB	AB	BILE DUCT PROLIFERATION	AB
17-8496	SK	P	CANALICULAR	DUCTULAR PROLIFERATION NOTED	AB
17-8495	JS	P	BILE PLUGS	AB	AB
17-8664	HS	AB	AB	AB	AB
17-8732	SK	AB	BILE PLUGS	AB	AB
17-9013	MGP	AB	AB	AB	P
17-9239	HT	P	INTRACYTOPLASMIC	AB	P
17-9361	HS	AB	AB	BILE DUCT PROLIFERATION	AB
17-9710	JS	AB	AB	AB	AB
17-9820	V	AB	AB	AB	AB
17-10174	RS	AB	AB	DUCTOPENIA	AB
17-10377	SP	AB	AB	AB	AB
17-10474	UR	P	AB	AB	P
17-10502	SKG	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-10504	HKK	AB	AB	AB	AB
17-10537	SS	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-1936	ND	AB	AB	AB	AB

HISTO NO	INITIALS	HEPATOCYTES
16-9315	SD	MODERATE BALLOONING DEGENERTION
16-9793	M	MILD BALLONING DEGENERATION
16-10131	GS	INTRACYTOPLASMIC COPPER PIGMENTS
16-10668	DS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-11131	AA	NP
16-11127	GS	BALLOONING DEGENERATION
16-11701	GS	HEPATOCYTES ROSETTING NOTED
16-11965	RL	MILD BALLOONING DEGENRATION
16-12253	VKA	MILD BALLONING DEGENERATION
16-12407	RD	NP
16-12459	JR	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
16-12560	GS	MILD BALLONING DEGENERATION
16-12508	DR	MODERATE BALLOONING DEGENERTION
'16-12826	AR	GIANT CELL TRANSFORMATION NOTED
16-13009	RK	HEPATOCYTE ROSETING NOTED
16-13132	BK	KUPFFER CELLS PROMINENCE
16-13091	RA	HEPATOCYTES ROSETING NOTED
17-03	RK	HEPATOCYTES ROSETTING NOTED
17-196	SS	FEATHERY DEGENERATION OF HEPATOCYTES ALONG WITH HEPATOCYTES ROSETTING
17-425	RS	HEPATOCYTES ROSETTING AND NEUTROPHILIC SATELLITOSIS SEEN
17-471	BK	MILD BALLONING DEGENERATION
17-534	PD	GIANT CELL TRANSFORMATION NOTED
17-775	NG	UR
17-1062	RK	MILD BALLOONING DEGERATION
17-1185	GC	EPITHELIOD CELL GRANULOMAS WITH LANGHANS AND FOREIGN BODY GIANT CELL REACTION.
17-1248	RSA	MODERATE BALLOONING DEGENRATION
17-1326	DS	MILD BALLOONING AND NEUTROPHILIC SATELLITOSIS NOTED
17-1382	KS	MILD BALLONING DEGENERATION
17-1417	PS	NP
17-1470	RK	MULTIPLE EPITHELIOD CELL GRANULOMAS IN THE PARENCHYMA. GRANULOMAS ARE SMALL AND REGULAR FEW OF THEM ARE FIBROSING WITH AN INDISTINCT LYMPHOCYTIC COLLAR.
17-1623	NM	MILD BALLONING DEGENRATION
17-1700	VG	MILD BALLOONING AND NEUTROPHILIC SATELLITOSIS NOTED
17-1856	M	MILD BALLONING DEGENERATION
17-1966	PD	BALLOONING DEGENERATION
17-2239	BS	MILD INFILRATION BY CHRONIC INFLAMMATORY CELLS
17-2397	HS	MILD BALLONING DEGENRATION
17-2508	AK	HEPATOCYTES ROSETTING IDENTIFIED
17-2617	KL	NP
17-2615	IG	MILD BALLONING DEGENRATION
17-2683	AS	MILD BALLOONING AND HEPATOCYTES ROSETTING AND NEUTROPHILIC SATELLITOSIS SEEN
17-2981	DD	NP
17-3005	SPK	MILD BALLONING DEGENRATION
17-3041	AJ	TINY GRANULOMAS NOTED
17-3132	MS	MILD BALLONING DEGENRATION
17-3336	A	WELL FORMED EPITHELIOD GRANULOMAS
17-3456	HK	MILD BALLONING DEGENRATION
17-4152	SR	FORMATION OF HEPATOCYTIC ROSETTING NOTED
17-4567	SP	MODERATE BALLOONING AND NEUTROPHILIC SATELLITOSIS
17-4676	ML	ROSETTING NOTED
17-4738	N	GROUND GLASS HEPATOCYTES NOTED
17-4995	J	MILD BALLOONING DEGENERATION
17-5095	RK	MILD BALLOONING AND NEUTROPHILIC SATELLITOSIS
17-5253	DR	NP
17-5497	KK	EMPERIPOLESIS NOTED AND HEPATOCYTIC ROSSETTING
17-5723	VK	MANY EPITHELIOD CELL GRANULOMAS WITH LANGHANS TYPE GIANT CELLS
17-5928	RK	MILD BALLONING DEGENERATION
17-6520	B	MODERATE BALLOONING DEGENERTION
'17-6825	SK	MILD BALLONING DEGENRATION
17-6962	AK	CHOLENGITIS PRESENT
17-7018	AS	MILD BALLONING DEGENERATION
17-7338	MK	WELL FORMED EPITHELIOD GRANULOMAS
17-8000	BS	MILD BALLONING DEGENERATION
17-8496	SK	MODERATE BALLOONING DEGENERTION
17-8495	JS	MILD BALLONING DEGENERATION
17-8664	HS	MILD BALLONING DEGENERATION
17-8732	SK	MILD BALLONING DEGENERATION
17-9013	MGP	MILD BALLONING DEGENERATION
17-9239	HT	MILD BALLOONING
17-9361	HS	PORTAL SCAR SHOW DENSE INFILTRATION BY INFLAMMATORY CELLS SEEN AS LYMPHOCYTES, NEUTROPHILS AND FEW EOSINOPHILS
17-9710	JS	MODERATE BALLOONING DEGENERTION
17-9820	V	MILD BALLONING DEGENERATION
17-10174	RS	MILD BALLONING DEGENERATION
17-10377	SP	MILD BALLONING DEGENERATION OF HEPATOCYTES AND GLYCOGENIZATION OF HEPATOCYTES NUCLEI SEEN
17-10474	UR	MODERATE BALLOONING DEGENERATION
17-10502	SKG	MILD BALLONING DEGENERATION
17-10504	HKK	MILD BALLONING DEGENERATION
17-10537	SS	MILD BALLONING DEGENERATION
17-1936	ND	MILD BALLONING DEGENERATION

HISTO NO	INITIALS	INFLAMMATORY INFILTRATE PORTAL TRACTS/SEPTA/SINUSOIDS	PLASMA CELL RICH	GRANULOMA	INTERFACE HEPATITIS	SPOTTY HEPATIC NECROSIS
16-9315	SD	NO INFILTRATE	AB	AB	AB	AB
16-9793	M	MINIMAL CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
16-10131	GS	NOT IDENTIFIED	AB	AB	AB	AB
16-10668	DS	MILD INFILTRATION BY LYMPHOCYTES, HISTIOCYTES WITH INCREASED PORTAL FIBROSIS	AB	AB	AB	AB
16-11131	AA	MINIMAL INFILLTRATION BY LYMPHOCYTES	AB	AB	AB	P< 1 focus/10X
16-11127	GS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	AB
16-11701	GS	MODERATE MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	P
16-11965	RL	MILD ACUTE AND CHRONIC INFILTRATE	AB	AB	AB	AB
16-12253	VKA	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
16-12407	RD	MILD CHRONIC LYMPHOMONONUCLEAR CELL INFILTRATE	AB	AB	AB	P < 1/10X
16-12459	JR	INFLAMMATORY INFILTRATE ALONG WITH LYMPHOCYTES, HISTIOCYTES, FEW NEUTROPHILS AND OCCASIONAL EOSINOPHILS	AB	AB	AB	AB
16-12560	GS	NO INFILTRATE	AB	AB	AB	P
16-12508	DR	UNREMARKABLE	AB	AB	AB	AB
'16-12826	AR	MODERATE MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, LYMPHOCYTES AND EOSINOPHILS	AB	AB	AB	AB
16-13009	RK	MODERATE DEGREE OF INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HOSTIOCYTES, FEW PLASMA CELLS, EOSINOPHILS AND OCCASIONAL LYMPHOID AGGREGATE	AB	AB	P	AB
16-13132	BK	UNREMARKABLE	AB	AB	AB	P
16-13091	RA	MODERATE TO MARKED CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, PLASMA CELLS ,FEW EOSINOPHILS AND HISTIOCYTES	AB	AB	AB	AB
17-03	RK	MODERATE DEGREE OF MIXED INFLAMMATORY INFILTRATE ALONG WITH NEUTROPHILS, LYMPHOCYTES AND EOSINOPHILS	AB	AB	AB	AB
17-196	SS	MILD CHRONIC INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
17-425	RS	MODERATE INFILTRATE SEEN AS NEUTROPHILS, FEW LYMPHOCYTES AND MACROPHAGES	AB	AB	AB	AB
17-471	BK	MIXED INFLAMMATORY INFILTRATE ALONG WITH NEUTROPHILS- CHOLANGITIS	AB	AB	AB	AB
17-534	PD	MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES AND FEW HISTIOCYTES	AB	AB	P	AB
17-775	NG	MIXED INFLAMMATORY INFILTRATE ALONG WITH NEUTROPHILS, LYMPHOCYTES, EOSINOPHILS AND FEW HISTIOCYTES	AB	AB	P	AB
17-1062	RK	MINIMAL INFILTRATE	AB	AB	AB	P
17-1185	GC	NO INFILTRATE	AB	P	AB	AB
17-1248	RSA	DENSE ACUTE AND CHRONIC INFLAMMATORY INFILTRATE IN PORTAL TRACTS	AB	AB	AB	AB
17-1326	DS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
17-1382	KS	MILD EXPANSION AND INFILTRATION OF MILD CRONIC INFLAMMATORY CELLS	AB	AB	AB	P
17-1417	PS	NO INFILTRATE	AB	AB	AB	P
17-1470	RK	ABSENT	AB	P	AB	P
17-1623	NM	INFILTRATION BY PREDOMINANTLY CHRONIC INFLAMMATORY CELLS SEEN AS LYMPHOCYTES AND FEW PLASMA CELLS	AB	AB	P	P
17-1700	VG	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELLS SEEN AS LYMPHOCYTES, FEW PLASMA CELLS AND EOSINOPHILS NOTED	AB	AB	AB	AB
17-1856	M	MODERATE INFILTRATION BY PREDOMINANTLY CHRONIC INFLAMMATORY INFILTRATE ALONG WITH LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
17-1966	PD	MILD INFILTRATION BY MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW NEUTROPHILS, EOSINOPHILS, PLASMA CELLS AND OCCASIONAL HISTIOCYTES	AB	AB	AB	P
17-2239	BS	UNREMARKABLE	AB	AB	AB	AB
17-2397	HS	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS SEEN AS LYMPHOCYTES, FEW HISTIOCYTES AND PLASMA CELLS	AB	AB	P	AB
17-2508	AK	MIXED INFLAMMATORY INFILTRATE ALONG WITH LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL NEUTROPHILS AND PLASMA CELLS	AB	AB	AB	P
17-2617	KL	MILD INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	P
17-2615	IG	MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW NEUTROPHILS, EOSINOPHILS AND PLASMA CELLS AND HISTIOCYTES	AB	AB	P	P
17-2683	AS	MODERATE MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL EOSINOPHILS	AB	AB	AB	AB
17-2981	DD	MILD INFILTRATED BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL EOSIONOPHILS	AB	AB	P	P
17-3005	SPK	DENSE INFLAMMATORY INFILTRATE MAINLY LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
17-3041	AJ	INFILTRATED BY LYMPHOCYTES AND HISTIOCYTES	AB	P	AB	P
17-3132	MS	INFILTRATED BY MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
17-3336	A	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	P	AB	P
17-3456	HK	MODERATE TO MARKED MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES AND HISTOCYTES	AB	AB	P	P
17-4152	SR	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELLS WITH PRESENCE OF LYMPHOID AGGREGATE AND OCCASIONAL PLASMA CELLS	AB	AB	AB	P
17-4567	SP	MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, NEUTROPHILS AND OCCASIONAL EOSINOPHILS AND PLASMA CELLS	AB	AB	AB	P
17-4676	ML	EXPANDED AND INFILTRATED BY MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, NEUTROPHILS, FEW HISTIOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	AB	P
17-4738	N	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
17-4995	J	MILD MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES OCCASIONAL NEUTROPHILS AND PLASMA CELLS	AB	AB	P	AB
17-5095	RK	DENSE MIXED INFLAMMATORY INFILTRATE	AB	AB	P	P
17-5253	DR	MODERATE DEGREE OF LYMPHOMONONUCLEAR CELLS INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL EOSINOPHILS	AB	AB	AB	P
17-5497	KK	INFILTRATION BY MODERATE DEGREE OF INFLAMMATORY INFILTRATE COPOSED OF LYMPHOCYTES, HISTIOCYTES AND MANY PLASMA CELLS	AB	AB	P	P
17-5723	VK	MILD CHRONIC INFLAMMTORY INFILTRATES	AB	P	AB	P
17-5928	RK	MINIMAL CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
17-6520	B	MODERATE MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	P	AB
'17-6825	SK	MODERATE MIXED INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES, NEUTROPHILS AND PLASMA CELLS	AB	AB	P	P
17-6962	AK	DENSE MIXED INFLAMMATORY INFILTRATESEEN AS LYMPHOCYTES, MANY PLASMA CELLS, FEW NEUTROPHILS AND HISTIOCYTES	AB	AB	P	P
17-7018	AS	MODERATE MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, LYMPHOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
17-7338	MK	EXPANDED AND SHOW MILD INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES AND OCCASIONAL EOSINOPHILS	AB	P	AB	AB
17-8000	BS	MILD MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL EOSINOPHILS	AB	AB	AB	AB
17-8496	SK	MILD LYMPHOMONONUCLEAR INFILTRATE	AB	AB	AB	AB
17-8495	JS	UNREMARKABLE	AB	AB	AB	P
17-8664	HS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	P	P
17-8732	SK	UNREMARKABLE	AB	AB	P	AB
17-9013	MGP	MILD ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	P	P
17-9239	HT	NO PORTAL TRACTS SEEN	AB	AB	AB	AB
17-9361	HS	MILD CHRONIC INFLAMMTORY INFILTRATES	AB	AB	P	P
17-9710	JS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
17-9820	V	EXPANDED AND INFILTRATED BY ACUTE AND CHRONIC INFLAMMATORY CELLS	AB	AB	P	AB
17-10174	RS	MILD TO MODERATE DEGREE OF LYMPHOCYTIC INFILTRATE	AB	AB	AB	AB
17-10377	SP	MODERATE DEGREE OF CHRONIC INFLAMMATORY INFILTRATE COMPRISING OF LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
17-10474	UR	MILD DEGREE OF MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES AND NEUTROPHILS	AB	AB	AB	AB
17-10502	SKG	MODERATE MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, HISTIOCYTES, EOSINOPHILS AND OCCASIONAL PLASMA CELLS	AB	AB	P	AB
17-10504	HKK	MODERATE DEGREE OF INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES, OCCASIONAL NEUTROPHILS AND PLASMA CELLS	AB	AB	AB	AB
17-10537	SS	INFILTRATION BY MODERATE DEGREE OF INFLAMMATORY INFILTRATE SEEN AS HISTIOCYTES, LYMPHOCYTES AND NEUTROPHILS	AB	AB	P	AB
17-1936	ND	MILD INFILTRATION BY NEUTROPHILS AND LYMPHOCYTES	AB	AB	AB	AB

HISTO NO	INITIALS	CONFLUENT NECROSIS	FIBROSIS	STAINS		
				IRON	MASSON TRICHOME	RETICULIN
16-9315	SD	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERIVESCULAR AND PERICELLULAR FIBROSIS WITH INCREASED PORTAL FIBROSIS	MAINTAINED
16-9793	M	AB	AB	NEGATIVE	NO SIGNIFICANT INCREASE IN FIBROSIS	MAINTAINED
16-10131	GS	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERIVENULAR AND PERICELLULAR FIBROSIS	MAINTAINED
16-10668	DS	AB	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS	MAINTAINED
16-11131	AA	AB	AB	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS	MAINTAINED
16-11127	GS	AB	GRADE II	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	MAINTAINED
16-11701	GS	AB	GARDE II	NEGATIVE	HIGHLIGHTS PORTAL AND PERICELULAR FIBROSIS	MAINTAINED
16-11965	RL	P	GRADE IV	NEGATIVE	INCREASED FIBROSIS AND NODULE FORMATION	MAINTAINED
16-12253	VKA	AB	GRADE I	NEGATIVE	HIGHLIGHTS INCREASED PERICELLULAR FIBROSIS	MAINTAINED
16-12407	RD	AB	GRADE I	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	MAINTAINED
16-12459	JR	AB	GRADE II	PRESENCE OF HEMOSIDERIN IN KUPFFER CELLS	INCREASED FIBROSIS IN AN OCCASIONAL PORTAL TRACT	MAINTAINED
16-12560	GS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
16-12508	DR	AB	GRADE I	NEGATIVE	PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
'16-12826	AR	AB	AB	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS IN SCAR TISSUE	MAINTAINED
16-13009	RK	AB	AB	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS SEPTAL WITH NODULE FORMATION	MAINTAINED
16-13132	BK	P	GRADE II	NEGATIVE	HIGHLIGHTS PERICELLULAR FIBROSIS	MAINTAINED
16-13091	RA	AB	AB	NEGATIVE	HIGHLIGHTS SIGNIFICANT INCREASE IN FIBROSIS	MAINTAINED
17-03	RK	P	GRADE1	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
17-196	SS	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL AND PERIPORTAL FIBROSIS	MAINTAINED
17-425	RS	AB	GRADE III	NEGATIVE	HIGHLIGHTS PERIVENULAR AND PERICELLULAR FIBROSIS AND PORTAL SCAR	MAINTAINED
17-471	BK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION WITH FOCAL PERICELLULAR FIBROSIS	MAINTAINED
17-534	PD	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS AND ARCHITECTURAL DISTORTION	COLLAPSE
17-775	NG	AB	AB	NEGATIVE	DO NOTHIGHLIGHTS NODULE FORMATION AND INCREASED PERIPORTAL AND PERISEPTAL FIBROSIS	MAINTAINED
17-1062	RK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS PERICELLULAR FIBROSIS AND SCAR TISSUE	MAINTAINED
17-1185	GC	AB	AB	ND	NOT DONE	ND
17-1248	RSA	AB	GRADE III	NEGATIVE	HIGHLIGHT INCREASE PERICELLULAR AND PERISINOSOIDAL FIBROSIS	MAINTAINED
17-1326	DS	AB	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS	MAINTAINED
17-1382	KS	AB	AB	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
17-1417	PS	AB	GRADE II	NEGATIVE	HIGHLY INCREASED PORTAL FIBROSIS WITH PORTAL TO PORTAL FIBROSIS	MAINTAINED
17-1470	RK	AB	AB	NEGATIVE	NOT DONE	HIGHLIGHTED AROUND THE GRANULOMA
17-1623	NM	P	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS WITH ATTEMPTED NODULE FORMATION	MAINTAINED
17-1700	VG	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-1856	M	AB	GRADE IV	NEGATIVE	HIGHLIGHTS INCREASED PORTAL AND PERIPORTAL FIBROSIS	MAINTAINED
17-1966	PD	P	AB	NEGATIVE	HIGHLIGHTS FOCAL INCREASE IN PORTALL FIBROSIS	MAINTAINED
17-2239	BS	AB	GRADE I	NEGATIVE	HIGHLIGHTS PERIVENULAR AND PERIPORTAL FIBROSIS	MAINTAINED
17-2397	HS	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS FIBROSIS	MAINTAINED
17-2508	AK	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
17-2617	KL	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS FIBROSIS	MAINTAINED
17-2615	IG	P	GRADE II	NEGATIVE	HIGHLIGHTS FOCAL INCREASE IN PORTAL FIBROSIS	MAINTAINED
17-2683	AS	AB	GRADE IV	POSITIVE IN KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-2981	DD	AB	GRADE IV	NEGATIVE	HIGHLIGHTS FIBROSIS AND NODULE FORMATION	MAINTAINED
17-3005	SPK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
17-3041	AJ	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS AND BRIDGING SEPTAL	MAINTAINED
17-3132	MS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS FIBROSIS WITH NODULE FORMATION	MAINTAINED
17-3336	A	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	DOES NOT ACCENTUATE AROUND GRANULOMA
17-3456	HK	AB	GRADE I	NEGATIVE	HIGHLIGHTS FIBROSIS AND INCREASED PORTAL FIBROSIS	MAINTAINED
17-4152	SR	P	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-4567	SP	AB	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
17-4676	ML	AB	GRADE IV	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS AND ATTEMPTED NODULE FORMATION	FOCAL LOSS OF RETICULIN FRAMEWORK
17-4738	N	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
17-4995	J	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND PERICELLULAR FIBROSIS	MAINTAINED
17-5095	RK	AB	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
17-5253	DR	AB	GRADE IV	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
17-5497	KK	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
17-5723	VK	AB	GRADE I	NEGATIVE	ND	ND
17-5928	RK	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
17-6520	B	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS AND NODULE FORMATION	MAINTAINED
'17-6825	SK	AB	GRADE II	NEGATIVE	HIGHLIGHT THIN PORTAL SEPTAL INCREASED PERICELLULAR FIBROSIS AND ARCHITECTURAL DISTORTION	MAINTAINED
17-6962	AK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-7018	AS	AB	GRADE III	NEGATIVE	HGHLIGHT PERICELLULAR FIBROSIS WITH ATTEMPTED NODULE FORMATION	MAINTAINED
17-7338	MK	AB	GRADE II	HIGHLIGHTS IRON WITHIN KUPFFER CELLS	HIGHLIGHTS INCREASED PPORTAL FIBROSIS AND PORTOPORTAL SEPTAL	FOCAL COLLAPSE
17-8000	BS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-8496	SK	AB	GRADE IV	NEGATIVE	HIGLIGHTS PORTAL SCAR WITH INCREASED FIBROSIS	MAINTAINED
17-8495	JS	AB	GRADE II	FOCAL POSITIVITY IN KUPFFER CELLS	HIGHLIGHTS FIBROSIS	MAINTAINED
17-8664	HS	AB	GRADE I	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS WITH OCCASIONAL PORTAL PORTAL SEPTAL	MAINTAINED
17-8732	SK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS FIBROSIS AND NODULE FORMATION	RETICULIN CONDENSATION IN PORTAL SCARS
17-9013	MGP	AB	GRADE IV	NEGATIVE	HIGHLIGHTS FIBROSIS WITH PERICELLULAR ACCENTUATION	MAINTAINED
17-9239	HT	P	GRADE II	NEGATIVE	HIGHLIGHTS PROMINENT ACUTE ALCOHOLIC HEPATITIS	MAINTAINED
17-9361	HS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS SCAR WITH ATTEMPTED NODULE FORMATION	MAINTAINED
17-9710	JS	AB	GRADE II	NEGATIVE	HIGHLIGHTS FOCAL PERIVENULAR FIBROSIS WITH PORTAL FIBROSIS AND OCCASIONAL BUDDING FIBROSIS	MAINTAINED
17-9820	V	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-10174	RS	AB	GRADE II	INCREASED IRON IN KUPFFER CELLS	HIGHLIGHTS PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
17-10377	SP	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-10474	UR	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND PERIVENULAR AND PERICELLULAR FIBROSIS	MAINTAINED
17-10502	SKG	AB	GRADE I	FOCAL IRON DEPOSITS IN KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-10504	HKK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS PORTO PORTAL SEPTAL AND PERICELLULAR FIBROSIS	MAINTAINED
17-10537	SS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-1936	ND	AB	GRADE IV	NEGATIVE	HIGHLIGHTS BROAD PORTAL SCAR	MAINTAINED

HISTO NO	INITIALS	OTHERS
16-9315	SD	ND
16-9793	M	ND
16-10131	GS	RHODANINE FOR COPPER IS POSITIVE
16-10668	DS	ND
16-11131	AA	ND
16-11127	GS	ND
16-11701	GS	ND
16-11965	RL	ND
16-12253	VKA	ND
16-12407	RD	ND
16-12459	JR	ND
16-12560	GS	ND
16-12508	DR	ND
'16-12826	AR	ND
16-13009	RK	ND
16-13132	BK	ND
16-13091	RA	ND
17-03	RK	ND
17-196	SS	ND
17-425	RS	ND
17-471	BK	ND
17-534	PD	ND
17-775	NG	ND
17-1062	RK	ND
17-1185	GC	ND
17-1248	RSA	ND
17-1326	DS	ND
17-1382	KS	ND
17-1417	PS	ND
17-1470	RK	20% ZN FOR AFB IS NEGATIVE
17-1623	NM	ND
17-1700	VG	ND
17-1856	M	ND
17-1966	PD	ND
17-2239	BS	ND
17-2397	HS	ND
17-2508	AK	ND
17-2617	KL	ND
17-2615	IG	ND
17-2683	AS	ND
17-2981	DD	ND
17-3005	SPK	ND
17-3041	AJ	20% ZN STAIN FOR AFB IS NEGATIVE
17-3132	MS	ND
17-3336	A	20% ZN STAIN FOR AFB IS NEGATIVE
17-3456	HK	RHODANINE FOR COPPER AND COPPER ASSOCIATED PROTEIN IS NEGATIVE
17-4152	SR	ND
17-4567	SP	ND
17-4676	ML	ND
17-4738	N	ND
17-4995	J	ORCEIN AND RHODENINE FOR COPPER AND COPPER ASSOCIATED PROTEIN SHOW FOCAL POSITIVITY IN PERISEPTAL AREAS
17-5095	RK	ND
17-5253	DR	ND
17-5497	KK	ND
17-5723	VK	20% ZN STAIN FOR AFB IS NEGATIVE
17-5928	RK	ND
17-6520	B	ND
'17-6825	SK	ND
17-6962	AK	ND
17-7018	AS	ND
17-7338	MK	ND
17-8000	BS	ND
17-8496	SK	ND
17-8495	JS	ND
17-8664	HS	ND
17-8732	SK	ND
17-9013	MGP	ND
17-9239	HT	ND
17-9361	HS	ND
17-9710	JS	ND
17-9820	V	ND
17-10174	RS	ND
17-10377	SP	ND
17-10474	UR	ND
17-10502	SKG	ND
17-10504	HKK	ND
17-10537	SS	ND
17-1936	ND	ND

HISTO NO	INITIALS	IMPRESSION
16-9315	SD	NASH GRADE 5 STAGE 2
16-9793	M	NASH GRADE 4 STAGE 0
16-10131	GS	NASH GRADE 1 STAGE 0
16-10668	DS	PRIMARY SCLEROSING CHOLANGITIS
16-11131	AA	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1 STAGE 0
16-11127	GS	CHRONIC VIRAL HEPATITIS(HCV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 2, STAGE 1
16-11701	GS	PRIMARY SCLEROSING CHOLANGITIS
16-11965	RL	ASH GRADE 3 STAGE 4
16-12253	VKA	NASH GRADE 4 STAGE 1
16-12407	RD	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2 GRADE 1, STAGE 1
16-12459	JR	CIRRHOSIS- IDIOPATHIC
16-12560	GS	NASH GRADE 1 STAGE 0
16-12508	DR	NASH GRADE 5 STAGE 1
'16-12826	AR	CHRONIC HEPATITIS- IDIOPATHIC
16-13009	RK	AUTOIMMUNE HEPATITIS
16-13132	BK	DILI (HERBAL MEDICINES)
16-13091	RA	AUTOIMMUNE HEPATITIS
17-03	RK	CHOLESTATIC LIVER DISEASE
17-196	SS	AUTOIMMUNE HEPATITIS
17-425	RS	ASH GRADE 3 STAGE 4
17-471	BK	NASH GARDE 3 STAGE 4
17-534	PD	AUTOIMMUNE HEPATITIS
17-775	NG	NO SPECIFIC PATHOLOGY
17-1062	RK	ASH GRADE 3 STAGE 4
17-1185	GC	GRANULOMATOUS INFLAMMATION- SARCOIDOSIS
17-1248	RSA	ASH GRADE 6 STAGE 3
17-1326	DS	ASH GRADE 5 STAGE 3
17-1382	KS	NASH GRADE 2 STAGE 0
17-1417	PS	CHRONIC VIRAL HEPATITIS WITH MILD ACTIVITY(HBV POSITIVE) HAI SCORE 4, GRADE 2 STAGE 2.
17-1470	RK	GRANULOMATOUS INFLAMMATION-SARCOIDOSIS
17-1623	NM	PRIMARY SCLEROSING CHOLANGITIS
17-1700	VG	ASH GRADE 6 STAGE 4
17-1856	M	NASH GRADE 6 STAGE 4
17-1966	PD	ACUTE HEPATITIS (HAV RELATED)
17-2239	BS	ASH GRADE 3 STAGE 1
17-2397	HS	AUTOIMMUNE HEPATITIS
17-2508	AK	AUTOIMMUNE HEPATITIS
17-2617	KL	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2 GRADE 1, STAGE 0
17-2615	IG	ACUTE HEPATITIS (HAV POSITIVE)
17-2683	AS	ASH GRADE 6 STAGE 4
17-2981	DD	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MILD ACTIVITY HAI SCORE 5 GRADE 2 STAGE 4
17-3005	SPK	AUTOIMMUNE HEPATITIS
17-3041	AJ	GRANULOMATOUS INFLAMMATION WITH ARCHITECTURE DISTORTION-SARCOIDOSIS
17-3132	MS	PRIMARY SCLEROSING CHOLANGITIS
17-3336	A	GRANULOMATOUS HEPATITIS- TUBERCULOSIS
17-3456	HK	AUTOIMMUNE HEPATITIS
17-4152	SR	AUTOIMMUNE HEPATITIS
17-4567	SP	ASH GRADE 7 STAGE 4
17-4676	ML	LARGE DUCT OBSTRUCTION
17-4738	N	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) HAI SCORE 2 GRADE I STAGE I
17-4995	J	NASH GRADE 6 STAGE 4
17-5095	RK	ASH GRADE 7 STAGE 4
17-5253	DR	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4 , GRADE 2 STAGE 1
17-5497	KK	AUTOIMMUNE HEPATITIS
17-5723	VK	GRANULOMATOUS HEPATITIS-TUBERCULOSIS
17-5928	RK	NASH GRADE 1 STAGE 2
17-6520	B	NASH GRADE 6 STAGE 2
'17-6825	SK	AUTOIMMUNE HEPATITIS
17-6962	AK	AUTOIMMUNE HEPATITIS
17-7018	AS	NASH GRADE 3 STAGE 2
17-7338	MK	GRANULOMATOUS HEPATITIS-SARCOIDOSIS
17-8000	BS	NASH GRADE 4 STAGE 4
17-8496	SK	NASH GRADE 3 STAGE 3
17-8495	JS	DILI (ATT)
17-8664	HS	CHRONIC HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 6 GRADE 2 STAGE 3
17-8732	SK	CIRRHOSIS- IDIOPATHIC
17-9013	MGP	NASH GRADE 4 STAGE 4
17-9239	HT	ASH GRADE 1 STAGE 1
17-9361	HS	CIRRHOSIS – IDIOPATHIC
17-9710	JS	NASH GRADE 3 STAGE 2
17-9820	V	NASH GRADE 4 STAGE 4
17-10174	RS	PRIMARY BILIARY CIRRHOSIS
17-10377	SP	NASH GRADE 4 STAGE 2
17-10474	UR	NASH GRADE 5 STAGE 4
17-10502	SKG	NASH GRADE 5 STAGE 1
17-10504	HKK	NASH GRADE 4 STAGE 3
17-10537	SS	CIRRHOSIS- IDIOPATHIC
17-1936	ND	CIRRHOSIS- IDIOPATHIC

				HISTORY									BIOCHEMISTRY								
HISTO NO	INITIALS	MRD	AGE/SEX	JAUNDICE	DIABETES	ALCOHAL	OBESITY	ABDOMINAL MASS	HEPATO MEGALY	SPLENOME GALY	LYMPHADENO PATHY	RADIOLOGICAL FINDINGS	BILI TOTAL	BILI DIRECT	SGOT	SGPT	ALBUMIN	ALP	PROTIENS	VIRAL MARKERS	AUTOIMMUNE MARKERS
17-10544	AC	344125	68/M	P	AB	AB	AB	AB	AB	AB	AB	ND	3	2.5	50	55	5	80	6.5	NR	ND
17-10548	AL	324858	36/M	P	AB	AB	P	AB	P	AB	AB	HEPATOMEGALY	3.6	3.02	30	26	3	611	7.6	NR	ND
17-10615	VM	342995	56/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.52	0.28	37	25	4.24	127	7.8	NR	ND
17-10660	G	346395	35/M	AB	AB	AB	AB	AB	P	AB	AB	ND	1	0.2	42	45	5	80	7	NR	ND
17-10663	MS	346165	35/M	AB	P	AB	AB	AB	P	AB	AB	ND	1	0.2	50	55	3.6	100	8	NR	ND
17-10722	IK	81090	63/F	P	AB	AB	AB	AB	P	P	AB	ND	1.9	0.33	39	17	4.17	70	7.6	NR	ANA1+,ASMA 1+
17-10862	GS	345713	35/M	P	AB	AB	AB	AB	P	AB	AB	HEPATOMEGALY	7.37	4.66	51	33	2.4	128	6.4	NR	ANA 1+
17-11090	NR	144383	55/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.11	0.42	37	24	3.5	150	5.8	NR	ANCA 1+
17-11338	SS	353104	20/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.23	1.01	49	40	3.2	430	6.8	NR	ND
17-11522	HS	357400	48/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	4	3.5	75	80	4.5	180	7.2	NR	ND
17-11665	RK	87401	46/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.3	0.2	15	22	4	92	8	HEP B DNA (PCR)-2.38	ND
17-11667	GK	330394	55/F	AB	AB	AB	AB	AB	P	AB	AB	ND	0.24	0.09	33	23	4.5	66	8.2	NR	ND
17-11676	UK	328168	60/F	AB	P	AB	AB	AB	P	AB	AB	ND	0.52	0.25	50	28	4.2	95	8	NR	NEGATIVE
17-11753	MA	348774	13/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.42	0.22	15	16	1.8	131	5.2	NR	ND
17-11767	SS	282178	55/F	P	P	AB	P	AB	P	P	AB	HEPATOSPLEENOMEGALY	2.3	2	46	54	3.5	107	7.5	NR	ND
17-11806	PK	364684	31/M	AB	AB	AB	P	AB	P	AB	AB	HEPATOMEGALY	1	0.9	50	55	5	100	7	NR	ND
17-12136	JP	209901	57/M	P	AB	AB	AB	AB	AB	AB	AB	ND	6	5.83	26	26	2.2	1507	6.2	NR	ANA 1+
17-12138	RS	358520	42/M	AB	AB	P	AB	AB	P	P	AB	ND	0.74	0.42	22	17	3.7	79	7.3	NR	ANA 1+
'17-12145	B	365822	45/M	AB	AB	AB	AB	AB	AB	P	AB	ND	4.75	4.2	101	60	3.76	96	7.8	ANTI HCV-REACTIVE	ANA 1+
17-12154	KK	367510	24/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.4	0.08	26	39	4.26	178	8.4	NR	ND
17-12262	SS	361114	48/M	P	AB	AB	AB	AB	AB	AB	AB	ND	16.1	13.7	214	71	2.5	166	5.1	NR	NEGATIVE
17-12314	SK	299123	51/F	AB	AB	P	P	AB	P	P	AB	ND	0.37	0.14	66	101	4.5	94	8.1	NR	NEGATIVE
17-12569	AP	372795	48/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	0.43	0.25	40	42	2	100	5.6	NR	ND
17-12628	UK	370196	51/F	P	AB	AB	AB	AB	AB	AB	AB	CHOLESTATIC LIVER DISEASE	22.14	18.3	316	132	2.4	145	4.9	NR	NEGATIVE
17-12801	MSB	118847	20/M	AB	AB	AB	AB	AB	P	AB	AB	HEPATOMEGALY	0.8	0.4	55	103	4	100	3	HEP B DNA (PCR)-1.06	ND
17-12884	GK	382355	55/F	P	P	AB	P	AB	AB	P	AB	SPLEENOMEGALY	0.79	0.14	36	30	4.2	99	7.4	NR	ND
17-12915	GS	322668	40/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.9	1	35	40	5	120	7	HEP-B POSITIVE	ND
17-13079	RS	27175	35/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.69	0.29	24	22	4.3	152	7.1	HEP-B POSITIVE	ND
17-13315	SK	337157	54/F	P	AB	AB	AB	AB	AB	AB	AB	ND	4.14	2.09	85	78	2.56	249	5.1	NR	NEGATIVE
17-13435	MS	353974	60/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.8	0.3	45	69	5.2	101	6	NR	ND
17-13619	BS	391223	34/M	AB	AB	P	AB	AB	P	P	AB	CIRRHOSIS	19.2	16.1	95	37	2.6	131	6.8	NR	ND
17-14185	PK	400273	65/M	P	AB	AB	AB	AB	AB	AB	AB	ND	1.12	0.43	9	11	3.8	45	6.4	NR	ND
17-14437	U	523678	53/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	8	7.5	35	40	4	100	8	NR	ND
17-14557	VS	213807	53/F	AB	AB	AB	AB	AB	AB	AB	AB	GRANULOMATOUS INFLAMMATION	0.7	0.28	28	33	4.2	163	7.1	NR	ND
18-1650	SS	434678	24/M	P	AB	AB	AB	AB	AB	AB	AB	ND	0.73	0.21	46	27	4.7	90	8	HEP B POSITIVE	NEGATIVE
18-3036	MS	461413	48/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	15.2	12.5	50	55	5	120	6.5	NR	NEGATIVE
18-3292	LR	450984	72/M	P	AB	AB	AB	AB	P	P	AB	CIRRHOSIS	16.47	26.4	45	50	3.5	60	6	HCV-POSITIVE	NEGATIVE
18-3707	KLB	468003	70/M	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.84	0.53	32	8	1.6	264	8.2	NR	NEGATIVE
18-3771	SD	468745	70/F	AB	AB	AB	AB	AB	AB	P	AB	CIRRHOSIS	0.77	0.4	44	29	3.4	87	5.7	NR	NEGATIVE
18-3839	SK	412917	43/M	P	AB	AB	AB	AB	AB	AB	AB	CHOLESTATIC LIVER DISEASE	10.82	6.86	211	104	3.08	586	4.3	NR	NEGATIVE
18-3888	SM	471118	52/F	P	P	AB	AB	AB	AB	P	AB	ND	14.4	21.4	110	45	3.5	155	6.2	NR	NEGATIVE
18-4238	DV	463658	60/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS FATTY LIVER GRADE 3	1.19	0.81	261	247	2.9	314	9.5	NR	NEGATIVE
18-4483	NR	469559	59/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.5	0.54	107	69	3.2	146	6.5	NR	ND
18-4505	SD	479044	42/F	AB	AB	AB	AB	AB	AB	AB	AB	HEPATIC SOL	0.59	1.53	16	22	2.3	423	ND	NR	ND
18-4638	NK	481237	60/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	0.51	0.11	29	26	4.27	150	7.2	NR	NEGATIVE
18-4653	KK	58071	50/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.69	0.47	72	66	3.6	202	6.7	NR	ND
18-4849	DK	483784	52/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	1.63	1.13	67	63	3.6	367	7.1	NR	ANA 3+ SMA1+ LKM 1+
18-4878	PK	366642	27/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.2	5	31	53	ND	124	ND	HBV-POSITIVE	ND
18-5233	SD	488630	58/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.79	0.41	82	50	2.6	320	7.7	NR	NEGATIVE
18-5474	SS	490484	22/M	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	0.86	0.44	32	35	3.4	131	6.8	HCV- POSITIVE	NEGATIVE
18-5495	MK	489851	43/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.67	0.24	210	90	4.1	60	6.6	NR	NEGATIVE
18-5577	SK	484815	32/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	1.46	0.98	61	32	3.8	377	9.2	NR	ANA 2+, ASMA 2+,LKM 1+, ANCA 2+
18-5611	RK	494321	55/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	14.5	16.6	194	97	4.2	113	8.5	NR	NEGATIVE
18-5703	DK	496356	57/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.69	3.43	290	121	2.5	338	7.8	NR	ANA-2+ ASMA-3+ AMA- 1+
18-5744	JS	492884	70/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	13.6	15.8	138	79	2.5	951	5.4	NR	ND
18-5852	KL	496481	57/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	20.9	16.2	95	54	2.7	219	7.1	NR	ANA 1+, ANCA 1+ LKM-NEG
18-5857	RD	497734	65/M	AB	P	AB	AB	AB	AB	AB	AB	ND	0.62	0.2	48	40	4.2	223	7.8	NR	NEGATIVE
18-5935	A	501768	29/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	3.5	5.5	42	45	3.6	140	6.5	NR	ND
18-6090	P	401503	28/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1	0.52	51	46	3.9	163	8.4	NR	ANA3+, AMA2+, ANCA 2+
18-6092	GK	470320	45/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.37	0.22	272	184	4.3	111	8.6	NR	NEGATIVE
18-6153	SS	245059	62/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.97	0.26	190	110	3.9	69	6.5	HBV POSITIVE	NEGATIVE
18-6182	J	499333	29/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.22	0.18	495	482	4.1	137	8.4	NR	ANA2+,ASMA-3+,LKM-2+,ANCA-2+
18-6417	HB	491194	44/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.02	0.39	51	24	2.6	84	6.7	NR	NEGATIVE
18-6688	SK	487869	52/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.6	0.15	43	43	2.97	114	7.3	NR	NEGATIVE
18-6768	RK	196288	52/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	2.18	1.04	92	31	2.8	88	7.3	NR	ANA-1+, ASMA 2+
18-6862	MS	478230	66/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.83	0.53	129	147	3.5	436	6.2	ND	ND
18-6899	STD	513805	50/M	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	28.7	24.3	500	530	2.2	476	4.1	ND	ND
18-7045	HK	517142	35/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.24	0.5	36	34	6.4	218	8	ND	ND
18-7075	RR	513169	22/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC HEPATITIS	2.6	1.2	866	771	3.6	214	7.1	NR	ASMA-1+, ANA-3+ LKM 2+
18- 7237	YK	491150	48/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	8.88	7.39	506	1492	2.7	786	4.1	ND	ND
18-8272	GK	470320	45/F	AB	AB	AB	AB	AB	AB	AB	AB	FATTY LIVER	0.37	0.22	272	184	4.3	111	4.3	NR	LKM-1+, ASMA-1+, AMA-1+,
18-8395	SR	535249	45/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.22	1	154	152	3.6	220	5.8	NR	ND
19-8494	MK	755913	30/F	P	AB	AB	AB	AB	AB	AB	AB	ND	12.5	6.93	43	50	2.47	120	4.9	NR	ND
18-8538	R	509966	52/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.89	0.41	59	32	4	124	8.9	NR	ND
18-8577	RB	533140	45/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	26.6	18.3	146	118	5.2	90	5.4	NR	ND
18-8692	PS	363847	55/M	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	1.49	0.72	40	16	3.9	42	8.1	NR	ND
18-8805	KK	421302	39/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.13	0.51	113	890	2.5	111	9.1	NR	ND
18-9064	SD	542200	57/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.31	0.65	30	27	3.5	97	6.7	NR	ND

HISTO NO	INITIALS	CERULOPLASMIN	IRON	LIVER BIOPSY GROSS NUMBER	LENGTH	ARCHITECTURE	FATTY CHANGE TYPE	FATTY CHANGE DEGREE
17-10544	AC	ND	ND	1	1.5 CM	DISTORTED LOBULAR ARCHITECTURE WITH ATTEMPTED NODULE FORMATION. BROAD PORTAL SCAR ALSO SEEN	MACROVESICULAR	0.35
17-10548	AL	ND	ND	1	1.2 CM	LOBULAR DISARRAY	MACROVESICULAR	0.6
17-10615	VM	ND	ND	2	0.3 TO 0.5 CM	DISTORTED WITH NODULAR FORMATION	MACROVESICULAR	0.05
17-10660	G	ND	ND	3	0.1,0.4 & 0.5 CM	ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.8
17-10663	MS	ND	ND	1	1 CM	MAINTAINED ARCHITECTURE	MACROVESICULAR	0.25
17-10722	IK	21	ND	1	0.5 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.1
17-10862	GS	21.2	ND	1	1.2 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION SEPRATED BY THICK FIBROUS SEPTAL	AB	AB
17-11090	NR	32.4	57	1	1 CM	LOBULAR DISARRAY	AB	AB
17-11338	SS	ND	ND	2	1.2 & 1.2 CM	MAINTAINED	AB	AB
17-11522	HS	ND	ND	2	0.2 & 1CM	MAINTAINED	AB	AB
17-11665	RK	ND	ND	1	1 CM	MAINTAINED	MACROVESICULAR	0.05
17-11667	GK	25.1	70.4	1	0.4 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.5
17-11676	UK	25.7	ND	1	1.5 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.33
17-11753	MA	ND	38.8	1	1.2 CM	MAINTAINED	MACROVESICULAR	0.9
17-11767	SS	32.3	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	AB	AB
17-11806	PK	ND	ND	6	02 TO 0.5 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICKENED FIBROUS SEPTAL	MACROVESICULAR	0.05
17-12136	JP	ND	61.1	1	1.8 CM	MILD LOBULAR DISARRAY	AB	AB
17-12138	RS	ND	ND	1	2 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICKENED FIBROUS SEPTAL	MACROVESICULAR	0.05
'17-12145	B	18.6	ND	1	0.4 CM	NODULE FORMATION SEPRATED BY THICK FIBROUS SEPTAL	AB	AB
17-12154	KK	ND	ND	1	1.2 CM	MAINTAINED	AB	AB
17-12262	SS	32.4	ND	1	0.7 CM	DISTORTED	AB	AB
17-12314	SK	ND	ND	1	0.8 CM	MAINTAINED	MACROVESICULAR	0.6
17-12569	AP	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.2
17-12628	UK	24.5	ND	1	1.5CM	LOBULAR DISARRAY	AB	AB
17-12801	MSB	ND	ND	1	1.3 CM	MAINTAINED	AB	AB
17-12884	GK	ND	ND	2	0.4 & 0.5 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.4
17-12915	GS	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.1
17-13079	RS	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	AB	AB
17-13315	SK	18.3	ND	2	1 & 0.6 CM	DISTORTED WITH OCCASIONAL ATTEMPTED NODULE FORMATION	AB	AB
17-13435	MS	ND	ND	1	1.3 CM	MAINTAINED	MACROVESICULAR	0.66
17-13619	BS	29.1	82.2	3	0.8, 1 AND 1.5 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	>75%
17-14185	PK	ND	ND	1	1.3 CM	MAINTAINED ARCHITECTURE	AB	AB
17-14437	U	ND	ND	MULTIPLE	0.2 TO 0.5 CM	MAINTAINED	MACROVESICULAR	0.4
17-14557	VS	ND	ND	1	1.1 CM	MAINTAINED	AB	AB
18-1650	SS	ND	ND	1	2 CM	MAINTAINED	MACROVESICULAR	0.05
18-3036	MS	ND	ND	1	2 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICK FIBROSIS SEPTA	AB	AB
18-3292	LR	ND	ND	1	1.6 CM	DISTODRTED WITH OCCASIONAL ATTEMPTED NODULES FORMATION	AB	AB
18-3707	KLB	36.4	ND	3	0.3, 1.2, 1.3 CM	DISTODRTED WITH OCCASIONAL ATTEMPTED NODULES FORMATION	MACROVESICULAR	0.15
18-3771	SD	21.4	ND	1	1.3 CM	DISTODRTED WITH OCCASIONAL ATTEMPTED NODULES FORMATION	MACROVESICULAR	0.05
18-3839	SK	ND	ND	2	1.5,2 CM	MAINTAINED	AB	AB
18-3888	SM	ND	ND	1	1.2 CM	DISTORTED WITH OCCASIONAL ATTEMPTED NODULES FORMATION	AB	AB
18-4238	DV	ND	NEGATIVE	2	0.1,0.2 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
18-4483	NR	ND	ND	1	2 CM	FOCAL DISTORTION WITH NODULAR FORMATION	MACROVESICULAR	0.5
18-4505	SD	ND	ND	1	1.2 CM	MAINTAINED	HEPATOCYTES SHOW MILD BALLONING DEGENERATION	AB
18-4638	NK	ND	ND	1	1.0 CM	DISTORTED WITH THIN PORTO PORTEL SEPTA	MACROVESICULAR	0.2
18-4653	KK	ND	ND	3	0.2,0.3 & 1.6 CM	DISTORTED ARCHITECTURE WITH ATTEMPTED NODULE FORMATION	AB	AB
18-4849	DK	ND	ND	3	0.2,1,1.8 CM	DISTORTED WITH NODULE FORMATION	AB	AB
18-4878	PK	ND	ND	2	05, 0.5 CM	FOCAL ARCHITECTURAL DISTORTION WITH PORTO PORTALSEPTA	AB	AB
18-5233	SD	ND	ND	1	1.8 CM	DISTORTED	MACROVESICULAR	0.15
18-5474	SS	22.3	54.9	MULTIPLE	0.1 TO 0.4	DISTORTED WITH PORTO PORTEL SEPTA	AB	AB
18-5495	MK	ND	15.6	1	1.2 CM	MAINTAINED	AB	AB
18-5577	SK	34.4	10.8	1	1.5 CM	DISTORTED HEPATIC PARENCHYMA WITH NODULAR FORMATION	AB	AB
18-5611	RK	ND	58	2	1, 0.2 CM	MAINTAINED	MACROVESICULAR	0.5
18-5703	DK	ND	17.1	2	2 CM	DISTORTED WITH NODULAR FORMATIONSEPRATED BY VARIABLALY SIZED FIBROUS SEPTA	AB	AB
18-5744	JS	ND	ND	2	1, 1 CM	MAINTAINED ARCHICTECTURE	AB	AB
18-5852	KL	44.5	ND	1	1.3 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
18-5857	RD	ND	45.9	2	1.8, 2.2 CM	DISTORTED WITH NODULAR FORMATION SEPRATED BY THIN TO THICK FIBROUS SEPTA	MACROVESICULAR	0.4
18-5935	A	ND	100.5	1	1.8 CM	LOBULAR DISARRAT WITH THIN PORTO PORTAL SEPTA PORTOCAVAL SEPTA	MACROVESICULAR	0.35
18-6090	P	24	227.3	2	0.1 TO 0.6 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION WITH FIBROUS THICK SEPTA	MACROVESICULAR	0.2
18-6092	GK	38	ND	2	0.1 TO 1 CM	MAINTAINED WITH FOCAL THIN PORTO PORTAL SEPTATE	MACROVESICULAR	0.05
18-6153	SS	ND	NEGATIVE	1	2.2 CM	MAINTAINED	AB	AB
18-6182	J	24.2	NEGATIVE	1	1.6 CM	LOBULAR DISARRAY OF HEPATIC PARENCHYMA	AB	AB
18-6417	HB	ND	18.6	1	1 CM	SHOWS SCAR TISSUE WITH SOLITARY NODULE	AB	AB
18-6688	SK	25.1	ND	3	0.1-0.2 CM	DISTORTED HEPATIC ARCHETURE WITH NODULE FORMATION	MACROVESICULAR	0.05
18-6768	RK	21.8	94.8	4	0.1 TO 0.2 CM	DISTORTED HEPATIC ARCHETURE	AB	AB
18-6862	MS	ND	13	1	1.5 CM	MAINTAINED WELL FORMED EPITHELIOID CELL GRANULOMAS IN LOBULAR PARENVHYMA	MACROVESICULAR	0.1
18-6899	STD	ND	58.9	1	2CM	MAINTAINED WITH SOME LOBULAR DISARRAY	AB	AB
18-7045	HK	ND	ND	1	1.2 CM	DISTORTED HEPATIC ARCHITECTURE WITH NODULE FORMATION SEPRATED BY THICK FIBROUS SEPTA	AB	AB
18-7075	RR	15.8	26.9	1	1.5 CM	MARKED LOBULAR DISARRAY OF HEPATIC PARENCHYMA	AB	AB
18- 7237	YK	ND	ND	2	1.8 AND 1.7 CM	MAINTAINED WITH LOBULAR DISARRY	AB	AB
18-8272	GK	38.3	ND	1	1.8 CM	FOCAL ARCHITECTURE DISTORTION PORTO PORTEL SEPTA	MACROVESICULAR	0.15
18-8395	SR	ND	NEGATIVE	1	1.3 CM	DISTORTED WITH NODULAR FORMATION	AB	AB
19-8494	MK	ND	ND	4	0.1,0.1,0.1,0.3 CM	DISTORTED PORTAL AND LOBULAR ACHITECTURE WITH NODULE FORMATION	AB	AB
18-8538	R	ND	NEGATIVE	1	1.2 CM	DISTORTED WITH LOBULAR FORMATION	AB	AB
18-8577	RB	ND	ND	1	0.4 CM	MAINTAINED	AB	AB
18-8692	PS	29.8	227.8	MULTIPLE	0.1 TO 0.2 CM	DISTORTED .TINY SOLITARY NODULE IDENTIFIED	AB	AB
18-8805	KK	ND	ND	3	0.1,0.2,0.4 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
18-9064	SD	18.5	17	1	0.2 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	P	P

HISTO NO	INITIALS	CHOLESTASIS	BILE	BILE DUCT	MALLORY DENK BODIES
17-10544	AC	AB	AB	AB	AB
17-10548	AL	AB	AB	AB	AB
17-10615	VM	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-10660	G	AB	BILE PLUGS	AB	AB
17-10663	MS	AB	AB	AB	AB
17-10722	IK	AB	AB	AB	P
17-10862	GS	AB	BILIRUBINOSTASIS WITH OCCASIONAL BILE PLUGS	AB	AB
17-11090	NR	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
17-11338	SS	AB	AB	AB	AB
17-11522	HS	AB	AB	AB	AB
17-11665	RK	PERIVENULAR	AB	AB	AB
17-11667	GK	AB	AB	AB	AB
17-11676	UK	AB	AB	AB	P
17-11753	MA	AB	AB	AB	AB
17-11767	SS	AB	AB	AB	P
17-11806	PK	FOCAL INTRACELLULAR BILIRUBINOSTASIS	BILE PLUGS	DUCTULAR PROLIFERATION NOTED	P
17-12136	JP	P	BILE PLUGS SEEN	AB	AB
17-12138	RS	AB	FOCAL	DUCTULAR PROLIFERATION NOTED	P
'17-12145	B	AB	AB	AB	AB
17-12154	KK	AB	AB	AB	AB
17-12262	SS	INTRAHEPATIC AND INTRA CANALICULAR CHOLESTASIS	AB	AB	AB
17-12314	SK	AB	AB	AB	AB
17-12569	AP	AB	AB	AB	AB
17-12628	UK	INTRACELLULAR AND CANALICULAR TYPE	AB	AB	AB
17-12801	MSB	AB	AB	AB	AB
17-12884	GK	AB	AB	AB	AB
17-12915	GS	AB	AB	AB	AB
17-13079	RS	AB	AB	AB	AB
17-13315	SK	AB	AB	AB	AB
17-13435	MS	AB	AB	AB	AB
17-13619	BS	INTRACANALICULAR	BILE PLUGS	BILE DUCT PROLIFERATION	P
17-14185	PK	AB	AB	AB	AB
17-14437	U	INTRAHEPATIC	BILE PLUGS	AB	P
17-14557	VS	AB	AB	AB	AB
18-1650	SS	AB	AB	AB	AB
18-3036	MS	AB	BILE PLUGS SEEN	DUCTULAR PROLIFERATION NOTED	P
18-3292	LR	P	AB	AB	P
18-3707	KLB	P	AB	AB	AB
18-3771	SD	AB	AB	AB	AB
18-3839	SK	P	AB	AB	AB
18-3888	SM	INTRACELLULAR CHOLESTASIS	AB	AB	AB
18-4238	DV	AB	AB	AB	AB
18-4483	NR	AB	AB	AB	AB
18-4505	SD	AB	AB	AB	AB
18-4638	NK	AB	AB	AB	AB
18-4653	KK	AB	MINIMAL INTRACELLULAR BILIRUBINESTASIS	AB	AB
18-4849	DK	P	AB	AB	AB
18-4878	PK	AB	AB	AB	AB
18-5233	SD	AB	AB	AB	AB
18-5474	SS	MILD INTRACELLULAR CHOLESTASIS	AB	AB	AB
18-5495	MK	AB	AB	AB	AB
18-5577	SK	AB	AB	AB	DILATED
18-5611	RK	INTRACELLULAR CHOLESTASIS	BILE PLUGS	AB	AB
18-5703	DK	INTRACELLULAR CHOLESTASIS	AB	DUCTULAR REACTION	P
18-5744	JS	INTRACELLULAR CHOLESTASIS	AB	AB	AB
18-5852	KL	INTRACELLULAR CHOLESTASIS	BILE PLUGS	CHOLESTASIS	AB
18-5857	RD	AB	AB	AB	AB
18-5935	A	AB	AB	AB	AB
18-6090	P	AB	AB	AB	AB
18-6092	GK	AB	AB	AB	AB
18-6153	SS	AB	AB	AB	AB
18-6182	J	AB	AB	AB	AB
18-6417	HB	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
18-6688	SK	AB	AB	AB	AB
18-6768	RK	INTRACELLULAR CHOLESTASIS	AB	DUCTULAR PROLIFERATION NOTED	AB
18-6862	MS	AB	AB	AB	AB
18-6899	STD	CANALICULAR	BILE PLUGS	AB	P
18-7045	HK	AB	AB	AB	AB
18-7075	RR	AB	AB	AB	AB
18- 7237	YK	CENTRILOBULAR BILIRUBINOSTASIS	AB	CUFFED AND INFILTRATED BY LYMPHOCYTES INFILTRATE	AB
18-8272	GK	AB	AB	AB	AB
18-8395	SR	AB	AB	AB	AB
19-8494	MK	CANALICULAR	BILE PLUGS	AB	AB
18-8538	R	AB	AB	AB	AB
18-8577	RB	AB	AB	AB	AB
18-8692	PS	AB	AB	AB	AB
18-8805	KK	AB	AB	PROLIFERATION	AB
18-9064	SD	P	AB	AB	AB

HISTO NO	INITIALS	HEPATOCYTES
17-10544	AC	MODERATE BALLOONING DEGENERATION
17-10548	AL	MILD BALLONING DEGENERATION
17-10615	VM	MILD BALLONING DEGENERATION
17-10660	G	MILD INFILTRATES AND NEUTROPHILIC SATELLITOSIS
17-10663	MS	MILD BALLOONING DEGENERATION
17-10722	IK	MILD BALLOONING DEGENERATION
17-10862	GS	MODERATE BALLOONING DEGENERATION
17-11090	NR	MILD BALLONING DEGENERATION
17-11338	SS	WELL FORMED EPITHELIOD GRANULOMAS
17-11522	HS	PINKISH ACELLULAR DISARRAY
17-11665	RK	NP
17-11667	GK	MILD BALLOONING DEGENERATION
17-11676	UK	MILD BALLOONING DEGENERATION
17-11753	MA	MODERATE BALLOONING DEGENRATION
17-11767	SS	MODERATE BALLOONING DEGNERATION
17-11806	PK	MILD BALLOONING DEGENERATION
17-12136	JP	CHOLANGITIS
17-12138	RS	MILD BALLOONING DEGENERATION
'17-12145	B	NP
17-12154	KK	UNREMARKABLE
17-12262	SS	MODERATE BALLOONING DEGENRATION
17-12314	SK	MILD BALLOONING DEGENERATION
17-12569	AP	MILD BALLOONING DEGENERATION
17-12628	UK	MILD BALLONING DEGENERATION
17-12801	MSB	NP
17-12884	GK	MILD BALLONING DEGENERATION
17-12915	GS	BALLOONING DEGENERATION
17-13079	RS	NP
17-13315	SK	MILD BALLOONING DEGENERATION
17-13435	MS	MILD BALLOONING DEGENERATION
17-13619	BS	MODERATE BALLOONING DEGENRATION
17-14185	PK	MILD BALLOONING DEGENRATION
17-14437	U	MILD BALLOONING DEGENRATION
17-14557	VS	WELL FORMED EPITHELIOD GRANULOMAS
18-1650	SS	NP
18-3036	MS	EMPERIPOLESIS
18-3292	LR	NP
18-3707	KLB	MILD BALLOONING DEGENERATION
18-3771	SD	MILD BALLOONING DEGENERATION
18-3839	SK	MILD BALLOONING DEGENRATION
18-3888	SM	MILD BALLOONING DEGENRATION
18-4238	DV	MILD BALLOONING DEGENERATION
18-4483	NR	MILD BALLOONING DEGENRATION
18-4505	SD	UR
18-4638	NK	MILD BALLOONING DEGENERATION
18-4653	KK	MILD BALLOONING DEGENRATION
18-4849	DK	EMPERIPOLESIS AND HEPATOCYTIC ROSSETTING
18-4878	PK	GROUND GLASS HEPATOCYTES
18-5233	SD	MODERATE BALLOONING DEGENRATION
18-5474	SS	NP
18-5495	MK	MILD BALLOONING DEGENRATION
18-5577	SK	MILD BALLOONING DEGENRATION
18-5611	RK	MILD BALLOONING DEGENRATION
18-5703	DK	EMPERIPOLESIS AND HEPATOCYTIC ROSSETTING
18-5744	JS	TINY HISTIOCYTIC COLLECTIONS
18-5852	KL	ROSETTE FORMATION.
18-5857	RD	MILD BALLOONING DEGENERATION
18-5935	A	MODERATE BALLOONING DEGENRATION
18-6090	P	MILD BALLOONING DEGENRATION
18-6092	GK	MILD BALLOONING DEGENERATION
18-6153	SS	NP
18-6182	J	MILD BALLOONING DEGENRATION
18-6417	HB	MILD BALLOONING DEGENRATION
18-6688	SK	MILD BALLOONING DEGENERATION
18-6768	RK	MILD BALLOONING DEGENRATION
18-6862	MS	MILD BALLOONING DEGENRATION
18-6899	STD	MILD BALLOONING DEGENRATION
18-7045	HK	MILD BALLOONING DEGENRATION
18-7075	RR	MILD BALLOONING DEGENRATION
18- 7237	YK	IN CENTRILOBULAR AREA PROMINENT PERIVENULAR HEPATOCYTE NECROSIS.
18-8272	GK	MILD BALLOONING DEGENERATION
18-8395	SR	EMPERIPOLESIS AND HEPATOCYTIC ROSSETTING
19-8494	MK	MILD BALLOONING DEGENRATION
18-8538	R	MILD BALLOONING DEGENRATION
18-8577	RB	MILD BALLOONING DEGENRATION
18-8692	PS	MODERATE BALLOONING DEGENRATION
18-8805	KK	MILD BALLOONING DEGENRATION
18-9064	SD	MILD BALLOONING DEGENRATION

HISTO NO	INITIALS	INFLAMMATORY INFILTRATE PORTAL TRACTS/SEPTA/SINUSOIDS	PLASMA CELL RICH	GRANULOMA	INTERFACE HEPATITIS	SPOTTY HEPATIC NECROSIS
17-10544	AC	MODERATE DEGREE OF MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS, LYMPHOCYTES AND FEW HISTIOCYTES	AB	AB	P	AB
17-10548	AL	MILD INFILTRATION BY MIXED INFLAMMATORY INFILTRATE COMPRISING LYMPHOCYTES, NEUTROPHILS, MACROPHGAES AND FEW EOSINOPHILS	AB	AB	AB	AB
17-10615	VM	MODERATE LYMPHOCYTIC INFILTRATE	AB	AB	P	AB
17-10660	G	MILD MIXED INFILTRATE COMPRISING OF LYMPHOCYTES, FEW HISTIOCYTES AND NEUTROPHILS	AB	AB	AB	AB
17-10663	MS	MINIMAL LYMPHOCYTES INFILTRATE WITH INCREASED PORTAL FIBROSIS, PERIPORTAL SPUR AND OCCASIONAL PORTO PORTAL SEPTAL	AB	AB	AB	AB
17-10722	IK	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
17-10862	GS	MILD DEGREE OF LYMPHOCYTES, INFILTRATE ALONG WITH NEUTROPHILS AND HISTIOCYTES	AB	AB	AB	AB
17-11090	NR	DENSE INFLAMMATORY INFILTRATE BY LYMPHOCYTES, HISTIOCYTES, FEW NEUTROPHILS, EOSINOPHILS AND PLASMA CELLS	AB	AB	P	P
17-11338	SS	UNREMARKABLE	AB	WITH LANGHANS AND	AB	AB
17-11522	HS	PINKISH ACELLULAR DISARRAY	AB	AB	AB	AB
17-11665	RK	MILD INFLAMMATORY INFILTRATE AND INCREASED FIBROSIS	AB	AB	AB	P
17-11667	GK	MILD INFILTRATION BY MIXED INFLAMMATORY INFILTRATE	AB	AB	P	P
17-11676	UK	MILD INFILTRATION BY MIXED INFILTRATE COMPRISING OF LYMPHOCYTES AND FEW NEUTROPHILS	AB	AB	P	P
17-11753	MA	ABSENT	AB	AB	AB	AB
17-11767	SS	MILD LYMPHOMONONUCLEAR INFILTRATE	AB	AB	AB	AB
17-11806	PK	MILD INFILTRATED BY LYMPHOCYTES, HISTIOCYTES, FEW NEUTROPHILS AND PLASMA CELLS	AB	AB	AB	P
17-12136	JP	MILD INFILTRATION BY MIXED INFILTRATE COMPOSED OF LYMPHOCYTES, NEUTROPHILS AND OCCASIONAL EOSINOPHILS	AB	AB	P	P
17-12138	RS	MODERATE DEGREE OF MIXED INFILTRATE COMPOSED OF LYMPHOCYTES, FEW HISTIOCYTES AND NEUTROPHILS	AB	AB	P	AB
'17-12145	B	MILD TO MODERATE MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, FEW HISTIOCYTES AND NEUTROPHILS	AB	AB	P	P
17-12154	KK	NO	PERIDUCTAL COLLECTIO	P	AB	AB
17-12262	SS	MILD INFILTRATION BY LYMPHOCYTES, FEW HISTIOCYTES AND NEUTROPHILS	AB	AB	AB	AB
17-12314	SK	ABSENT	AB	AB	AB	P
17-12569	AP	MILD INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND FEW NEUTROPHILS	AB	AB	P	P
17-12628	UK	MILD ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
17-12801	MSB	MILD CHRONIC INFLAMMATORY INFILTRATE COMPRISING MAINLY OF LYMPHOCYTES AND ICREASED FIBROSIS	AB	AB	AB	P
17-12884	GK	MIXED INFLAMMATORY INFILTRATE	AB	AB	AB	P
17-12915	GS	MILD INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND NEUTROPHILS	AB	AB	P	5/10
17-13079	RS	MILD INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES	AB	AB	P	P
17-13315	SK	MILD INFILTRATION BY LYMPHOCYTES, OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
17-13435	MS	INFILTRATION BY MILD CHRONIC INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
17-13619	BS	DENSE ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
17-14185	PK	MILD DEGREE OF MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS AND LYMPHOCYTES	AB	AB	AB	AB
17-14437	U	EXPANDED AND INFILTRATED BY CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	P
17-14557	VS	MILD ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	P	P
18-1650	SS	MILD INFLAMMATORY INFILTRATE	AB	AB	AB	P
18-3036	MS	INFILTRATED WITH MODERATE TO MARKED INFLAMMATORY INFILTRATE COMPRISING OF LYMPHOCYTES, HISTIOCYTES, EOSINOPHILS, FEW NEUTROPHILS AND PLASMA CELLS	AB	AB	P	P
18-3292	LR	MILD INFLAMMATORY INFILTRATE SEEN ALONG WITH NEUTROPHILIC SATELLITOSIS,LYMPHOCYTES,HISTIOCYTES AND NEUTROPHILS.	AB	AB	AB	AB
18-3707	KLB	MODERATE INFLAMMATORY INFILTRATE MAINLY LYMPHOCYTES , HISTIOCYTES AND HISTIOCYTES	AB	AB	P	P
18-3771	SD	MODERATE INFLAMMATORY INFILTRATE MAINLY LYMPHOCYTES , HISTIOCYTES AND HISTIOCYTES	AB	AB	P	P
18-3839	SK	MINIMAL INFILTRATES MAINLY LYMPHOCYTES	AB	AB	AB	AB
18-3888	SM	MILD INFILTRATION BY LYMPHOCYTES, NEUTROPHILS AND FEW HISTIOCYTES	AB	AB	P	AB
18-4238	DV	MODERATE DEGREE MAINLY LYMPHOCYTES, HISTIOCYTES, FEW EOSINOPHILS AND PLASMA CELLS	AB	AB	P	P
18-4483	NR	MODERATE DEGREE OF LYMPHONUCLEAR INFILTRATE. PORTAL FIBROSIS WITH PORTO PORTAL SEPTAL	AB	AB	AB	AB
18-4505	SD	FOCAL DILATED, MILD INFILTRATION BY INFLAMMATORY INFILTRATE MAINLY NEUTROPHILS, LYMPHOCYTESAND HISTIOCYTES.	AB	AB	AB	AB
18-4638	NK	MILD INFILTRATION BY BY LYMPHOCYTES, HISTIOCYTES OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
18-4653	KK	LOBULAR PARENCHYMA SHOWS MIXED INFLAMMATORY INFILTRATE COMPOSED OF NEUTROPHILS AND LYMPHOCYTES	AB	AB	P	AB
18-4849	DK	LYMPHOCYTES, NEUTROPHILS, PLASMA CELLS AND OCCASIONAL EOSINOPHILS	AB	AB	P	AB
18-4878	PK	MILD INFILTRATION BY LYMPHOCYTES	AB	AB	AB	P
18-5233	SD	DENSE INFLAMMATORY INFILTRATE COMPRISING LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL PLASMA CELLS.	AB	AB	P	P
18-5474	SS	MINIMAL INFLAMMATION BY LYMPHOCYTES	AB	AB	AB	AB
18-5495	MK	MODERATE DEGREE OF INFLAMMATORY INFILTRATE COMPRISING OF LYMPHOCYTES, FEW HISTIOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
18-5577	SK	MILD TO MODERATE DEGREE OF INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES AND PLASMA CELLS. PROMINENT ROSETTE FORMATION SEEN.	AB	AB	P	AB
18-5611	RK	MODERATE DEGREE OF MIXED INFLAMMATORY INFILTRATE WITH NEUTROPHILS, HISTIOCYTES, LYMPHOCYTES AND FEW PLASMA CELLS.	AB	AB	AB	AB
18-5703	DK	MODERATE DEGREE OF INFLAMMATORY INFILTRATE WITH LYMPHOCYTES, PLASMA CELLS AND NEUTROPHILS.	AB	AB	P	AB
18-5744	JS	MILD INCREASED FIBROSIS WITH MINIMAL TO MILD INFLTRATE BY LYMPHOCYTIC INFILTRATE	AB	AB	AB	AB
18-5852	KL	MODERATE MIXEDINFLAMMATORY INFILTRATE WITH NEUTROPHILS, AND FEW LYMPHOCYTES.	AB	AB	AB	AB
18-5857	RD	MODERATE DEGREE OF INFLAMMATORY INFILTRATE WITH LYMPHOCYTES AND FEW PLASMA CELLS	AB	AB	P	AB
18-5935	A	MODERATEINFILTRATION BY LYMPHOCYTES	AB	AB	AB	AB
18-6090	P	MODERATE DEGREE OF LYMPHOMONONUCLEAR CELL INFILTRATE WITH LYMPHOCYTES AND MANY PLASMA CELLS.	AB	AB	P	AB
18-6092	GK	MILD INFLAMMATION BY LYMPHOCYTES AND FEW NUTROPHILS	AB	AB	AB	AB
18-6153	SS	MILD INFILTRATION AND INCREASED FIBROSIS	AB	AB	AB	AB
18-6182	J	DENSE INFILTRATION BY LYMPHOCYTES, HISTIOCYTES, PLASMA CELLS AND EOSINOPHILS.	AB	AB	PROMINENT	AB
18-6417	HB	MILD CHRONIC INFLAMMATORY INFILTRATES	AB	AB	AB	AB
18-6688	SK	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	P	AB
18-6768	RK	PORTAL SEAR INFILTRATED BY MODERATE DEGREE OF INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTICYTES AND FEW PLASMA CELLS	AB	AB	AB	AB
18-6862	MS	LYMPHONUCLEAR NUCLEAR	AB	P	AB	AB
18-6899	STD	MILD INFILTRATION BY LYMPHOCYTES, HISTIOCYTES, PLASMA CELLS AND OCCASIONAL EOSINOPHILS.	AB	AB	P	P
18-7045	HK	DILATION LYMPHOMONONUCLEAR INFILTRATION	AB	AB	AB	AB
18-7075	RR	INFILTRATION BY LYMPHOCYTES, HISTIOCYTES, EOSINOPHILS AND PLASMA CELLS	AB	AB	P	P
18- 7237	YK	MIXED INFLAMMATORY INFILTRATE WITH LYMPHOCYTES, NEUTROPHILS AND EOSINOPHILS	AB	AB	P	P
18-8272	GK	MINIMAL INFILTRATION BY LYMPHOCYTES	AB	AB	AB	AB
18-8395	SR	DENSE INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, PLASMA CELLS AND FEW EOSINOPHILS	AB	AB	P	AB
19-8494	MK	MILD TO MODERATE INFILTRATION BY LYMPHOCYTES AND HISTIOCYTES	AB	AB	FOCAL SPILLOVERS	AB
18-8538	R	PORTAL SCAR SHOWING INFILTRATION BY LYMPHOCYTES AND OCCASIONAL PLASMA CELS	AB	AB	P	P
18-8577	RB	BROAD PORTAL SCAR SHOWING MODERATE INFILTRATION BY LYMPHOCYTES, HISTIOCYTES AND PLASMA CELLS	AB	AB	AB	AB
18-8692	PS	DENSE INFLAMMATORY INFILTRATE WITH LYMPHOID AGGREGATES	AB	AB	AB	AB
18-8805	KK	PORTAL SCAR WITH CHRONIC INFLAMMATORY INFILTRATE WITH LYMPHOID AGGREGATE	AB	AB	AB	AB
18-9064	SD	CHRONIC INFLAMMATORY INFILTRATE PORTAL FIBROSIS	AB	AB	AB	AB

HISTO NO	INITIALS	CONFLUENT NECROSIS	FIBROSIS	STAINS		
				IRON	MASSON TRICHOME	RETICULIN
17-10544	AC	AB	GRADE IV	NEGATIVE	HIGHLIGHTS INCREASED PORTAL, PERICELLULAR, PERIVESCULAR FIBROSIS AND ATTEMPTED NODULE FORMATION	MAINTAINED
17-10548	AL	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTOCANAL SEPTAL WITH INCREASED PERIVENULAR AND PERICELLULAR FIBROSIS	MAINTAINED
17-10615	VM	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION WITH FOCAL PERICELLULAR FIBROSIS	MAINTAINED
17-10660	G	AB	GRADE IV	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
17-10663	MS	AB	GRADE II	NEGATIVE	MILD INCREASED PORTAL FIBROSIS WITH OCCASIONAL PERIPORTAL DPUR AND OCCASIONAL PORTO PORTAL SEPTAL	MAINTAINED
17-10722	IK	AB	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-10862	GS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND FOCAL PERICELLULAR FOBROSIS	MAINTAINED
17-11090	NR	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
17-11338	SS	P	AB	POSITIVE	ND	ACCENTUATED AROUND GRANULOMA
17-11522	HS	AB	AB	ND	ND	ND
17-11665	RK	AB	GRADE I	NEGATIVE	INCREASED PORTAL FIBROSIS IS AN OCCASIONAL PORTAL TRACT	MAINTAINED
17-11667	GK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS PERICELLULAR AND BRIDGING FIBROSIS	MAINTAINED
17-11676	UK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION WITH FOCAL PERICELLULAR AND BRIDGING FIBROSIS	MAINTAINED
17-11753	MA	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
17-11767	SS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION POSSIBLY NASH	MAINTAINED
17-11806	PK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION, INCREASED PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
17-12136	JP	AB	GRADE II	FOCAL COLLAPSE	FOCAL INCREASE IN PORTAL FIBROSIS	MAINTAINED
17-12138	RS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	LOSS OF FORMATION IN NODULAR
'17-12145	B	AB	GRADE IV	FOCAL POSITIVITY IN HEMOSIDERIN IN HEPATOCYTES AT PERISEPTAL AREAS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-12154	KK	AB	AB	NEGATIVE	ND	MAINTAINED
17-12262	SS	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERCELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
17-12314	SK	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERICELLULAR, PERIVENULAR, OCCASIONAL THIN PERTO PORTAL SEPTAL	NORMAL HEPATIC PARENCHYMA
17-12569	AP	P	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION, SEPTAL, PORTO-PORTAL AND PERICELLULAR FIBROSIS	MAINTAINED
17-12628	UK	AB	GRADEI	NEGATIVE	HIGHLIGHTS FIBROSIS	MAINTAINED
17-12801	MSB	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
17-12884	GK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-12915	GS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-13079	RS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-13315	SK	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTO PORTAL SEPTAL, PERI[PORTAL FIBROSIS AND ATTEMPTED NODULE FORMATION	MAINTAINED
17-13435	MS	AB	GRADE II	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
17-13619	BS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
17-14185	PK	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
17-14437	U	AB	AB	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS WITH PERICELLULAR AND FOCAL PERIVENULAR	MAINTAINED
17-14557	VS	AB	AB	NEGATIVE	HIGHLIGHTS PORTAL AND PERIVENULAR FIBROSIS	MAINTAINED
18-1650	SS	AB	AB	NEGATIVE	HIGHLIGHTS NO FIBROSIS	MAINTAINED
18-3036	MS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-3292	LR	AB	P	FOCALLY POSITIVE	HIGHLIGHTS PERICELLULAR FIBROSIS, P-P SEPTAE AND ATTEMPTED NODULE FOEMATION	MAINTAINED
18-3707	KLB	P	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
18-3771	SD	P	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-3839	SK	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
18-3888	SM	AB	GRADE IV	NEGATIVE	HIGHLIGHT NODULE FORMATION	MAINTAINED
18-4238	DV	AB	GRADE IV	NEGATIVE	HIGHLIGHTS BROAD PORTAL SCAR WITH OCCASIONAL PERICELLULAR AND PERIVASCULAR	MAINTAINED
18-4483	NR	AB	GRADE III	NEGATIVE	PORTO PORTAL SEPTA WITH ATTEMPTED NODULE FORMATION AND PERICELLULAR FIBROSIS	MAINTAINED
18-4505	SD	AB	AB	NEGATIVE	DOES NOT SHOW INCREASED FIBROSIS	MAINTAINED
18-4638	NK	AB	GRADE III	NEGATIVE	PORTOPORTAL SEPTA ALONG WITH FOCAL PERICELLULAR AND PERIVENIULAR FIBROSIS	MAINTAINED
18-4653	KK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-4849	DK	AB	GRADE IV	NEGATIVE	PORTOPORTAL SEPTAL AND NODULE FORMATION	ND
18-4878	PK	AB	AB	NEGATIVE	HIGHLIGHTS PPORTO PORTAL SEPTAL	MAINTAINED
18-5233	SD	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	ND
18-5474	SS	AB	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	ND
18-5495	MK	AB	GRADE II	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	ND
18-5577	SK	AB	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	ND
18-5611	RK	AB	GRADE I	NEGATIVE	HIGHLIGHTS MILD INCRESED IN PERICELLULAR FIBROSIS	ND
18-5703	DK	AB	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION	ND
18-5744	JS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED FIROSIS	MAINTAINED
18-5852	KL	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	ND
18-5857	RD	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND FOCAL PERICELLULAR FIBROSIS	ND
18-5935	A	AB	GRADE II	FOCALLY POSITIVE IN KUPFFER CELLS	HIGHLIGHTS PORTO PORTAL AND PERICELLULAR FIBROSIS	MAINTAINED
18-6090	P	AB	GRADE IV	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	ND
18-6092	GK	AB	AB	NEGATIVE	HIGHLIGHTS THIN PORTO PORTEL AND FOCAL PERIVENULAR AND PERICELLULAR FIBROSIS.	ND
18-6153	SS	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	ND
18-6182	J	AB	AB	NEGATIVE	HIGHLIGHTS THIN PORTO PORTEL AND FOCAL PERIVENULAR AND FOCAL NAVEL SEPTAL.	Maintained
18-6417	HB	AB	GRADE IV	NEGATIVE	HIGHLIGHTS BROAD PORTAL SEAR	ND
18-6688	SK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND FOCAL PERICELLULAR FIBROSIS	MAINTAINED
18-6768	RK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS PERICELLULAR FIBROSIS AND PORTAL SEAR	ND
18-6862	MS	AB	AB	NEGATIVE	ND	ND
18-6899	STD	AB	GRADE III	FOCALLY POSITIVE	INCREASED PORTAL FIBROSIS WITH SOME PERIPORTAL SPUR FORMATION	ND
18-7045	HK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULES	MAINTAINED
18-7075	RR	AB	GRADE II	FOCALLY POSITIVE IN KUPFFER CELLS	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	FOCAL COLLAPSE
18- 7237	YK	AB	AB	ND	ND	ND
18-8272	GK	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS, PERIVENULAR AND PERICELLULAR FIBROSIS WITH PORTO SEPTAL	ND
18-8395	SR	AB	GRADE IV	ND	HIGHLIGHTS INCREASED FIBROSIS. NORMAL LOBULAR ARCHICTURE	ND
19-8494	MK	AB	GRADE IV	POSITIVITY IN FEW KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-8538	R	AB	GRADE IV	ND	HIGHLIGHTS NODULE FORMATION AND INCREASED FIBROSIS	ND
18-8577	RB	AB	AB	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	MAINTAINED
18-8692	PS	AB	GRADE II	NEGATIVE	SHOW PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
18-8805	KK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-9064	SD	AB	GRADE III	NEGATIVE	HIGHLIGHTS MILD INCREASE IN PORTAL FIBROSIS	MAINTAINED

HISTO NO	INITIALS	OTHERS
17-10544	AC	ND
17-10548	AL	ND
17-10615	VM	ND
17-10660	G	CONGO RED STAIN AND RHODANINE STAIN FOR COPPER IS NEGATIVE
17-10663	MS	ND
17-10722	IK	ND
17-10862	GS	ND
17-11090	NR	ND
17-11338	SS	ND
17-11522	HS	CONGO RED STAIN FOR AMYLOID IS POSITIVE
17-11665	RK	ND
17-11667	GK	ND
17-11676	UK	ND
17-11753	MA	ND
17-11767	SS	ND
17-11806	PK	ND
17-12136	JP	IRON -FOCAL POSITIVITY IN KUPFFER CELLS
17-12138	RS	ND
'17-12145	B	ND
17-12154	KK	20% ZN STAIN FOR AFB AND GMS STAIN FOR FUNGUS ARE NEGATIVE
17-12262	SS	ND
17-12314	SK	ND
17-12569	AP	ND
17-12628	UK	ND
17-12801	MSB	ND
17-12884	GK	ND
17-12915	GS	ND
17-13079	RS	ND
17-13315	SK	ND
17-13435	MS	ND
17-13619	BS	ND
17-14185	PK	ND
17-14437	U	ND
17-14557	VS	ND
18-1650	SS	ND
18-3036	MS	ND
18-3292	LR	ND
18-3707	KLB	ND
18-3771	SD	ND
18-3839	SK	RHODAMINE- NEG
18-3888	SM	ND
18-4238	DV	ND
18-4483	NR	ND
18-4505	SD	ND
18-4638	NK	ND
18-4653	KK	ND
18-4849	DK	ND
18-4878	PK	ND
18-5233	SD	ND
18-5474	SS	ND
18-5495	MK	ND
18-5577	SK	ND
18-5611	RK	ND
18-5703	DK	ND
18-5744	JS	ND
18-5852	KL	ND
18-5857	RD	ND
18-5935	A	ND
18-6090	P	ND
18-6092	GK	ND
18-6153	SS	ND
18-6182	J	ND
18-6417	HB	ND
18-6688	SK	ND
18-6768	RK	ND
18-6862	MS	ZN AFB IS NEGATIVE
18-6899	STD	ND
18-7045	HK	ND
18-7075	RR	ND
18- 7237	YK	ND
18-8272	GK	ND
18-8395	SR	ND
19-8494	MK	ND
18-8538	R	ND
18-8577	RB	ND
18-8692	PS	ND
18-8805	KK	ND
18-9064	SD	ND

HISTO NO	INITIALS	IMPRESSION
17-10544	AC	NASH GRADE 5 STAGE 4
17-10548	AL	NASH GRADE 4 STAGE 2
17-10615	VM	NASH GRADE 3 STAGE 4
17-10660	G	NASH GRADE 5 STAGE 4
17-10663	MS	NASH GRADE 2 DTAGE 1
17-10722	IK	NASH GRADE 3 STAGE 4
17-10862	GS	NASH GRADE 2 STAGE 4
17-11090	NR	PRIMARY SCLEROSING CHOLANGITIS
17-11338	SS	GRANULOMATOUS INFLAMMATION- SARCOIDOSIS
17-11522	HS	AMYLOIDOSIS
17-11665	RK	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 1, GRADE 1 STAGE 1
17-11667	GK	NASH GRADE 4 STAGE 4
17-11676	UK	NASH GRADE 3 STAGE 4
17-11753	MA	NASH GRADE STAGE 0
17-11767	SS	NASH GRADE 3 STAGE 4
17-11806	PK	NASH GRADE 3 STAGE 4
17-12136	JP	LARGE DUCT OBSTRUCTION
17-12138	RS	NASH GRADE 3 STAGE 4
'17-12145	B	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MILD ACTIVITY HAI SCORE 4 , GRADE 2, STAGE 2
17-12154	KK	GRANULOMATOUS INFLAMMATION - SARCOIDOSIS
17-12262	SS	NASH GRADE 2 STAGE 2
17-12314	SK	NASH GRADE 3 STAGE 3
17-12569	AP	NASH GRADE 3 STAGE 4
17-12628	UK	AUTOIMMUNE HEPATITIS
17-12801	MSB	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1 STAGE 1
17-12884	GK	NASH GRADE 4 STAGE 4
17-12915	GS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MODERATE ACTIVITY (HAI SCORE 9, GRADE 3 AND STAGE 4
17-13079	RS	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1 AND STAGE 4
17-13315	SK	NASH GRADE 3 STAGE 4
17-13435	MS	NASH GRADE 5 STAGE 0
17-13619	BS	ASH GRADE 7 STAGE 4
17-14185	PK	CHOLESTATIC LIVER DISEASE WITH CHOLANGITIS
17-14437	U	NASH GRADE 5 STAGE 4
17-14557	VS	GRANULOMATOUS INFLAMMATION- SARCOIDOSIS
18-1650	SS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2 GRADE 1 STAGE 0
18-3036	MS	LARGE DUCT OBSTRUCTION
18-3292	LR	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MILD ACTIVITY HAI SCORE 5 GRADE 2 STAGE 3
18-3707	KLB	NASH GRADE 3 STAGE 4
18-3771	SD	NASH GRADE 3 STAGE 4
18-3839	SK	LARGE DUCT OBSTRUCTION- SEPSIS INDUCED
18-3888	SM	AUTOIMMUNE HEPATITIS
18-4238	DV	NASH GRADE 3 STAGE 4
18-4483	NR	NASH GRADE 5 STAGE 3
18-4505	SD	NO SPECIFIC PATHOLOGY
18-4638	NK	NASH GRADE 2 STAGE 3
18-4653	KK	CIRRHOSIS- IDIOPATHIC
18-4849	DK	AUTOIMMUNE HEPATITS
18-4878	PK	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1, STAGE II
18-5233	SD	NASH GRADE 5 STAGE 4
18-5474	SS	CHRONIC VIRAL HEPATITIS (HCV POSITIVE), HAI SCORE I,GRADE I, STAGE III
18-5495	MK	CHRONIC HEPATITIS- IDIOPATHIC
18-5577	SK	AUTOIMMUNE HEPATITIS
18-5611	RK	CHOLESTATIC LIVER DISEASE
18-5703	DK	AUTOIMMUNE HEPATITIS
18-5744	JS	SARCOIDOSIS
18-5852	KL	NASH GRADE 5 STAGE 4
18-5857	RD	NASH GRADE 5 STAGE 4
18-5935	A	NASH GRADE 5 STAGE 3
18-6090	P	AUTOIMMUNE HEPATITIS
18-6092	GK	NASH GRADE 3 STAGE 2
18-6153	SS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 0, GRADE ,1 STAGE 1
18-6182	J	AUTOIMMUNE HEPATITIS
18-6417	HB	CIRRHOSIS- IDIOPATHIC
18-6688	SK	NASH GRADE 5 STAGE 4
18-6768	RK	AUTOIMMUNE HEPATITIS
18-6862	MS	GRANULOMATOUS HEPATITIS
18-6899	STD	DILI (AMOXICILLIN AND CLAVULINIC ACID)
18-7045	HK	CIRRHOSIS- IDIOPATHIC
18-7075	RR	AUTOIMMUNE HEPATITIS
18- 7237	YK	ACUTE CELLULAR REJECTION GRADE III WITH REJECTION ACTIVITY INDEX (RAI)- 7
18-8272	GK	NASH GRADE 3 STAGE 1
18-8395	SR	AUTOIMMUNE HEPATITIS
19-8494	MK	LARGE DUCT OBSTRUCTION
18-8538	R	CIRRHOSIS- IDIOPATHIC
18-8577	RB	CHRONIC HEPATITIS- IDIOPATHIC
18-8692	PS	NASH GRADE 3 STAGE 2
18-8805	KK	CIRRHOSIS- IDIOPATHIC
18-9064	SD	CHRONIC HEPATITIS- IDIOPATHIC

				HISTORY									BIOCHEMISTRY								
HISTO NO	INITIALS	MRD	AGE/SEX	JAUNDICE	DIABETES	ALCOHAL	OBESITY	ABDOMINAL MASS	HEPATO MEGALY	SPLENOME GALY	LYMPHADENO PATHY	RADIOLOGICAL FINDINGS	BILI TOTAL	BILI DIRECT	SGOT	SGPT	ALBUMIN	ALP	PROTIENS	VIRAL MARKERS	AUTOIMMUNE MARKERS
18-9250	HS	543693	60/M	AB	AB	P	AB	AB	AB	AB	AB	ND	4.62	4.17	95	58	3.2	269	6.3	NR	ND
18-10276	AK	560792	63/M	AB	AB	P	AB	AB	AB	AB	AB	CIRRHOSIS	2.19	1.34	29	46	3.2	85	7.1	NR	ND
18-10478	SV	563511	57/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.64	0.18	470	290	3.5	227	7.8	NR	ND
18-11287	DK	539844	50/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.94	0.27	68	32	3.36	104	7.7	NR	ND
18-11450	KS	567584	60/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.47	3.38	56	23	2.85	248	7	NR	ND
18-11967	RM	584546	26/M	P	AB	AB	AB	AB	AB	AB	AB	NAD	2.1	0.72	23	22	5.3	62	7.5	NR	ND
18-12298	GS	591261	58/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.72	0.29	56	82	4.5	710	7.9	HCV-REACTIVE	ND
18-12369	UR	593318	40/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.21	0.08	14	9	3.4	62	6.7	HCV-REACTIVE	ND
18-12464	JK	588863	41/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	3.4	2.09	245	169	1.8	222	10.8	NR	ANA 3+ AMA 2+ LKM 1+ ASMA 1+
18-12563	JB	595023	54/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	8.2	10.2	156	160	5.2	526	7.5	NR	ND
18-13610	HS	604424	51/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	4	1.97	102	79	3.5	461	9.8	HAV-POSITIVE	ND
18-13861	PS	604605	14/M	P	AB	AB	AB	AB	AB	AB	AB	ND	5.8	5.2	115	126	5.2	480	6.5	NR	ND
18-14352	MM	625535	25/F	AB	AB	AB	AB	AB	AB	P	AB	ND	8.5	7.6	142	153	4.6	265	7.5	NR	ASMA-2+, ANA 3+ LKM 1+
18-14363	NK	623202	38/F	AB	AB	AB	AB	AB	AB	AB	AB	FATTY LIVER GRADE 1	6.17	5.55	77	53	2.7	180	4.9	HAV-POSITIVE	ANA 1+
18-14706	PK	606332	53/F	P	AB	AB	AB	AB	AB	AB	AB	ND	4.2	3.6	40	24	4	79	9	NR	ANA IF 2+, ASMA 1+
18-14705	M	621882	42/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.6	0.2	33	27	4		7.3	NR	NEGATIVE
18-14954	AR	632013	50/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.5	1.2	40	40	3	120	5.4	NR	ND
18-14994	SR	617625	52/F	P	AB	AB	AB	AB	AB	P	AB	CIRRHOSIS	1.83	0.87	680	478	3.68	268	7.6	NR	ANA1+, ASMA 3+ LKM2+
18-15205	RS	614502	67/F	P	P	AB	AB	AB	AB	AB	AB	CIRRHOSIS	4.8	3.1	81	41	2.9	197	7.4	NR	ANA-3+, AMA-3+
18-15257	P	626293	53/M	P	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	2.22	1.56	66	66	2.7	101	5.6	NR	NEGATIVE
18-15395	RK	639354	60/M	P	AB	AB	AB	AB	AB	AB	AB	OBSTRUCTIVE BILIOPATHY	6.13	6	82	69	3	794	5.7	ANTI HBC-REACTIVE	ND
18-15404	AS	217649	49/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.36	0.19	57	75	3.9	1167	8.1	NR	ANA 1+
18-15545	AK	636598	40/F	P	AB	AB	AB	AB	AB	P	AB	CIRRHOSIS	8.18	7.54	311	202	3.6	121	7.3	NR	ANA 3+, SMA 3+
18-15559	KK	638310	58/F	AB	AB	AB	AB	AB	P	AB	AB	HETEROGENOUS ECHOTEXTURE	0.38	0.08	28	32	3.8	156	7.3	NR	ND
18-15709	SK	642810	55/M	AB	P	AB	AB	AB	AB	AB	AB	MULTIPLE LIVER AND SPLEENIC SOL	0.12	0.06	10	12	2.4	217	5.4	NR	ANA 1+
18-15734	DK	643881	53/F	AB	AB	AB	AB	AB	AB	AB	AB	GALL STONES	0.76	0.5	42	98	4.73	220	7.4	NR	ANA 1+
18-15818	LA	645184	41/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.3	0.09	40	50	5.2	100	7	HBV-POSITIVE	ND
19-52	SG	470162	63/F	AB	P	AB	AB	AB	AB	AB	AB	HEPATIC SOL	0.34	0.08	22	25	3.5	99	6.9	NR	ANA 4+, IgM ++
19-64	N	647151	17/F	AB	AB	AB	AB	AB	P	P	AB	HEPATOMEGALY WITHPORTAL HYPERTENSION	0.81	0.62	55	60	4.48	350	7.5	NR	ANA2+, ASMA3+, LKM1+,
19-190	KK	526444	31/F	P	AB	AB	AB	AB	P	AB	AB	CHRONIC LIVER DISEASE	27.1	22.16	129	49	2.7	445	7	NR	ANA-3+, AMA 1+, ASMA 1+
19-214	JS	647960	45/F	P	AB	P	AB	AB	P	P	AB	DECOMPENSATED CHRONIC LIVER DISAESE	7.03	11.9	74	38	3.3	500	7.1	NR	NEGATIVE
19-323	SS	651420	53/M	AB	AB	AB	AB	AB	AB	AB	AB	COARSE ECOTEXTURE	0.84	0.53	32	30	1.6	264	8.2	NR	NEGATIVE
19-334	AKB	649272	62/M	P	AB	AB	AB	AB	AB	P	AB	ND	1.17	0.61	38	30	2.6	215	7.1	HEP B POSITIVE	ANA1+
19-385	GS	643360	29/M	P	AB	P	AB	AB	AB	AB	AB	ND	11.4	9.39	48	37	3.7	404	7.1	NR	ND
19-639	SK	654079	32/F	P	AB	AB	AB	AB	AB	AB	AB	CLD WITH PORTAL HTN	0.62	0.23	57	220	1.7	784	7	NR	ND
19-726	RK	657614	53/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.79	0.64	14	25	2.7	42	5.6	HCV-REACTIVE	NEGATIVE
19-1099	RS	643368	55/M	AB	AB	AB	AB	AB	P	P	AB	CIRRHOSIS	1.36	1.02	50	55	4.2	182	6.2	NR	NEGATIVE
19-2040	K	650491	36/M	P	P	AB	AB	AB	P	AB	AB	ND	1.33	0.37	39	83	4.6	56	6.8	NR	NEGATIVE
19-2047	KS	666129	50/M	P	AB	AB	AB	AB	P	AB	AB	FATTY LIVER	0.78	0.26	32	51	4.6	72	6.9	NR	ND
19-2100	BS	652412	45/M	P	P	AB	AB	AB	AB	AB	AB	ND	10.2	9.5	144	109	3.5	825	8.5	HAV-POSITIVE	NEGATIVE
19-2086	JK	675437	50/F	AB	P	AB	AB	AB	AB	P	AB	CIRRHOSIS	2.5	2.1	150	162	4.2	250	7.2	NR	ANA-3+, IgG-4 TIMES RAISED
19-2191	MB	659898	60/F	P	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	0.57	0.38	22	39	4.4	454	7.7	NR	ANA2+, ASMA3+, LKM1+,
19-2257	TS	680185	60/M	P	AB	AB	AB	AB	AB	P	AB	CIRRHOSIS	0.85	0.53	32	35	1.6	264	8.2	NR	ND
19-2316	MK	680180	26/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	1	0.45	35	34	3.2	183	5.6	NR	ANA 2+ ASMA 3+ LKM 1+
19-2327	AS	633354	32/M	AB	AB	AB	AB	AB	AB	AB	AB	ILEAL STRICTURES	0.11	0.02	15	10	1.21	880	3.7	HCV POSITIVE	ND
19-2384	BS	616867	33/M	P	AB	AB	AB	AB	AB	AB	AB	SPLEENOMEGALY	12.8	11.51	141	113	3.9	117	9	HAV-POSITIVE	ASMA 1+, SLA-+,ANA 3+,LKM1+
19-2774	VA	620090	58/M	P	AB	AB	AB	AB	AB	AB	AB	OBSTRUCTIVE BILIOPATHY WITH SPLENIC VEIN THROMBOSIS	11.87	11.2	72	26	1.6	516	5.6	NR	ND
19-2813	M	674099	24/F	AB	AB	AB	AB	AB	P	P	AB	GALL STONES	1.6	0.5	60	57	2.5	131	8	NR	ND
19-2904	VA	51417	53/M	P	AB	AB	AB	AB	AB	AB	AB	ND	1	0.8	40	50	4	140	5.6	NR	ND
19-2923	PS	682334	46/M	AB	AB	AB	AB	AB	P	AB	AB	EARLY CLD	0.48	0.23	78	152	4.2	182	7.3	NR	NEGATIVE
19-3012	SM	689665	30/M	AB	P	AB	AB	AB	P	AB	AB	CHOLESTATIC LIVER DISEASE , MILD HEPATOMEGALY	8.2	8	80	87	4.2	114	7.8	NR	ND
19-3423	SK	610207	42/M	AB	AB	AB	AB	AB	AB	P	AB	NODULAR LIVER	1.2	1	62	65	3.2	125	6.5	NR	ND
19-3430	SK	694889	67/F	AB	AB	AB	AB	AB	AB	AB	AB	GALL STONES	2.4	1.5	52	54	3.5	142	6.2	NR	ND
19-5034	S	65521	72/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	3.5	3	62	70	2.5	235	5	NR	ND
19-5167	MMB	722306	72/M	AB	AB	AB	AB	AB	AB	P	AB	CIRRHOSIS	0.78	0.36	60	21	2.2	63	6.7	NR	ANA 1+, ASMA 3+
19-5170	SG	722474	57/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.62	0.63	16	22	3.8	74	6.4	NR	NEGATIVE
19-5177	BG	722273	63/F	P	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	1.24	0.72	30	10	3.2	90	9.4	NR	ASMA 3+, ANA2+, LKM 2+
19-5199	KK	722420	62/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.75	0.5	40	50	4.6	60	6.7	NR	ND
19-8185	KJ	265234	77/M	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	1.49	0.55	73	36	3.5	73	6.8	NR	ND
19-8229	TS	734417	54/M	AB	P	P	P	AB	AB	AB	AB	FIBROSIS-CIRRHOSIS	1.09	0.27	134	14	3.95	1190	6.8	NR	NEGATIVE
19-8284	BS	609286	52/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.44	0.07	53	75	4.05	85	6.7	HCV-REACTIVE	ND
19-8320	GG	761538	43/F	P	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	7.17	6.6	139	73	8.2	514	8.7	NR	AMA 3+ ASMA3+ LKM1+
19-8494	MK	755913	30/F	P	AB	AB	AB	AB	AB	AB	AB	ND	12.5	6.93	43	50	2.47	120	4.9	NR	ND
19-8556	S	700256	38/F	P	AB	AB	AB	AB	AB	AB	AB	ND	8.4	1.5	158	52	2.5	193	6.1	NR	ASMA & LKM 1+
19-8886	CD	727042	50/F	P	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	21.3	18.7	573	600	3.6	182	5.6	NR	ANA-4+ ASMA 2+ LKM1+
19-8907	RR	701258	62/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.2	5.6	103	72	2.5	506	4.5	HAV-POSITIVE	ND
19-9089	SS	751098	42/M	AB	AB	AB	P	AB	P	AB	AB	FATTY LIVER	0.45	0.18	510	610	4.2	114	7.2	NR	ND
19-9097	KD	770073	64/F	AB	AB	AB	AB	AB	P	P	AB	PORTAL HYPERTENSION	0.17	0.06	16	16	3.4	110	6.7	NR	ND
19-9209	AD	465935	43/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.54	0.63	30	20	4.3	810	8.3	NR	ND
18-9255	PS	772732	52/M	P	AB	P	AB	AB	AB	AB	AB	CIRRHOSIS	21.8	18.3	137	49	3.9	154	5.9	NR	ND
19-9391	MK	774735	44/M	P	AB	AB	AB	AB	AB	AB	AB	INTRAHEPTIC CHOLESTASIS AND MULTIPLE LIVER LESIONS	0.67	0.48	13	14	3.12	640	4.5	NR	ND
19-9430	JS	145878	41/M	AB	AB	AB	AB	AB	AB	AB	AB	FATTY LIVER	0.4	0.32	50	52	3.1	194	6.2	HBV-REACTIVE	ND
19-9472	MA	776694	48/M	P	AB	AB	AB	AB	AB	AB	AB	ND	9.9	8.9	24	13	2	464	5.5	NR	ND
19-9480	RK	747917	57/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.46	1.23	51	35	2.45	138	5.7	NR	ND
19-9503	N	773586	17/F	P	AB	AB	AB	AB	AB	AB	AB	ND	2.5	1.55	187	49	2.8	163	6.6	NR	ANTI SLA 1+
19-10485	YDS	792671	47/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.21	0.08	20	36	3.1	62	6	NR	ND
19-10535	HS	789311	59/M	P	P	P	AB	AB	AB	AB	AB	ND	22.8	18.5	78	23	3.7	116	5.8	NR	NEGATIVE
19-10889	KD	798773	57/F	P	AB	AB	AB	AB	AB	AB	AB	ND	1.38	1.01	82	90	2.47	166	7.2	NR	ANA 3+ ANCA 2+ ASMA 1+ LKM 1+

HISTO NO	INITIALS	CERULOPLASMIN	IRON	LIVER BIOPSY GROSS NUMBER	LENGTH	ARCHITECTURE	FATTY CHANGE TYPE	FATTY CHANGE DEGREE
18-9250	HS	28.9	ND	1	2 CM	DISTORTED WITH BLOOD PORTAL SCARS	MACROVESICULAR	0.75
18-10276	AK	23.7	80.2	1	1.2 CM	DISTORTED WITH NODULE FORMATION SEPERATED BY THICK FIBROUS SEPTA	MACROVESICULAR	0.3
18-10478	SV	ND	ND	1	1.2 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICK FIBROUS SEPTA	MACROVESICULAR	0.15
18-11287	DK	20.1	NEGATIVE	1	1.3 CM	DISTORTION OF PORTAL AND LOBULAR ARCHITECTURE WITH NODULE FORMATION BY THICK FIBROUS SEPTA	MACROVESICULAR	0.15
18-11450	KS	ND	NEGATIVE	1	1.5 CM	MAINTAINED	AB	AB
18-11967	RM	ND	NEGATIVE	1	1.2 CM	MAINTAINED. PT ARE UNREMARKABLE	AB	AB
18-12298	GS	ND	ND	1	1 1.8 CM	MAINTAINED	AB	AB
18-12369	UR	ND	NEGATIVE	1	1 CM	MAINTAINED ARCHITECTURE	AB	AB
18-12464	JK	ND	140.2	1	1.2CM	DISTORTED WITH NODULE FORMATION SEPERATED BY THICK FIBROUS SEPTA	AB	AB
18-12563	JB	ND	ND	1	1.5CM	MAINTAINED	AB	AB
18-13610	HS	ND	ND	2	0.8 CM	LOBULAR DISARRAY OF HEPATIC PARENCHYMA	AB	AB
18-13861	PS	ND	ND	1	1CM	MAINTAINED	AB	AB
18-14352	MM	39.4	7.5	1	1.4 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR STEATOSIS	0.05
18-14363	NK	ND	118.3	2	0.6 & 0.3 CM	BIOPSY SHOWS LOBULAR DISARRAY	MACROVESICULAR	0.05
18-14706	PK	ND	ND	1	1.5 CM	DISTORTED HEPATIC ARCHETURE WITH NODULE FORMATION	AB	AB
18-14705	M	31.6	43.5	1	1.8 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION	MACROVESCULAR STEATOSIS	0.1
18-14954	AR	ND	ND	1	1.5CM	DISTORTED	AB	AB
18-14994	SR	24.5	ND	1	1.8 CM	DISTORTED ARCHITECTURE WITH PRESENCE OF THIN TO THICK PORTAL SEPTA	AB	AB
18-15205	RS	20.5	ND	2	1.5 & 1.8 CM	DISTORTED WITH OCCASIONAL ATTEMPTED NODULE FORMATION	AB	AB
18-15257	P	ND	ND	1	1.4 CM	DISTORTED LOBULAR ARCHITECTURE WITH NODULE FORMATION	AB	AB
18-15395	RK	15.8	ND	1	1.5 CM	LOBULAR DISARRAY WITH INFILTRATION AND EXPANSION OF PORTAL TRACT	AB	AB
18-15404	AS	19.5		2	1.2 & 1.4 CM	MAINTAINED HEPATIC ARCHITECTURE MANY SMALL EPITHELIOID CELL GRANULOMAS NOTED IN LOBULAR PARENCHYMA	AB	AB
18-15545	AK	ND	76	1	1.7 CM	DISTORTED ARCHITECTURE	AB	AB
18-15559	KK	ND	ND	2	1.5 & 1.1 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION	MACROVESICULAR STEATOSIS	0.4
18-15709	SK	ND	ND	2	1.5 & 1.8 CM	LIVER BIOPSY SHOWS WELL FORMED EPITHELIOID CELL GRANULOMA WITHOUT LYMOPHOCYTIC CELLS	AB	AB
18-15734	DK	ND	ND	3	0.3,0.5,1.2 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	MACROVESICULAR STEATOSIS	0.15
18-15818	LA	ND	ND	1	1.8 CM	MAINTAINED ARCHICTECTURE	AB	AB
19-52	SG	NEGATIVE	NEGATIVE	2	1.2 & 1.5 CM	FOCAL ARCHITECTURAL DISTORTION WITH PORTO PORTAL SEPTA	AB	AB
19-64	N	ND	ND	2	1.4 & 1.5 CM	FOCAL DISTORTION OF HEPATIC AND LOBULAR ARCHITECTURE WITH MICRONODULE FORMATION IN PLACES	AB	AB
19-190	KK	ND	ND	3	0.1,0.5,0.8 CM	DISTORTED HEPATIC AND LOBULAR ARCHITECTURE WITH THIN TO THICK PORTAL NODULE FORMATION WITH SEPTA	AB	AB
19-214	JS	ND	ND	1	1.6 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION WITH PRESENCE OF PORTO PORTAL SEPTA	AB	AB
19-323	SS	15	ND	2	0.6 & 1.2 CM	MAINTAINED ARCHICTECTURE WITH MILD BALLORING DEGENERATION AND INCREASED FIBROSIS	MACROVESCULAR STEATOSIS	0.35
19-334	AKB	18	ND	2	0.5 & 1.5 CM	DISTORTED HEPATIC CHOLESTATIC WITH NODULE FORMATION	AB	AB
19-385	GS	ND	ND	1	1.2 CM	MAINTAINED ARCHITECTURE	MACROVESCULAR STEATOSIS	0.05
19-639	SK	ND	33	1	1.5 CM	DISTORTED HEPATIC ARCHITECTURE WITH NODULE FORMATION	AB	AB
19-726	RK	ND	ND	2	0.8 & 0.6 CM	MAINTAINED ARCHICTECTURE	MACROVESICULAR STEATOSIS	0.05
19-1099	RS	ND	ND	3	0.2,1.2,1.4	DISTORTED ARCHITECTURE	AB	AB
19-2040	K	17.4	ND			MAINTAINED ARCHITECTURE	MACROVESCULAR STEATOSIS	0.7
19-2047	KS	18.1	63.5	1	1.2 & 1.4 CM	MAINTAINED ARCHICTECTURE	MACROVESCULAR STEATOSIS	0.5
19-2100	BS	22	ND	1	1.3 CM	FOCAL ARCHITECTURAL DISTORTION WITH INCREASED PORTAL FIBROSIS WITH PORTOPORTAL SEPTA	AB	AB
19-2086	JK	ND	40.2	1	1.4 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION SEPRATED BY THICK FIBROUS SEPRAL	MACROVESCULAR STEATOSIS	0.1
19-2191	MB	ND	ND	6	0.1-1.5 CM	MAINTAINED HEPATIC ARCHICTECTURE	MACROVESCULAR STEATOSIS	0.05
19-2257	TS	ND	ND	1	1.5 CM	DISTORTED ARCHITECTURE WITH ATTEMPTED NODULE FORMATION SEPRATED BY VARIABLY SIZED SEPTA	MACROVESCULAR STEATOSIS	0.05
19-2316	MK	ND	93.2	1	1.7 CM	ARCHITECTURE DISTORTION WITH NODULE FORMATION	MACROVESCULAR STEATOSIS	0.05
19-2327	AS	ND	ND	1	2 CM	MAINTAINED HEPATIC ARCHITECTURE	AB	AB
19-2384	BS	ND	ND	1	1.3 CM	FOCAL ARCHITECTURED DISTORTION WITH INCREASED PORTAL FIBROSIS WITH PORTO PORTAL SEPTA	AB	AB
19-2774	VA	ND	41.5	MULTIPLE	0.1-0.5 CM	LOBULAR DISARRAY	AB	AB
19-2813	M	ND	ND	1	1.7 CM	MAINTAINED PORTAL ARCHITECTURE WITH MINIMAL LOBULAR DISARRAY	AB	AB
19-2904	VA	ND	ND	1		DISTORTED HEPATIC ARCHITECTURE WITH ATTEMPTED NODULE FORMATION SEPRATED BY THIN TO THICK FIBROUS SEPTA	AB	AB
19-2923	PS	ND	ND	1	0.8 CM	DISTORTED HEPATIC ARCHITECTURE WITH INCREASED PORTAL FIBROSIS WITH PORTO PORTAL SEPTA	MACROVESCULAR STEATOSIS	0.7
19-3012	SM	ND	ND	1	0.9 CM	MAINTAINED LOBULAR AND PORTAL ARCHITECTURE WITH PORTAL WITH OCCASIONAL PORTO PORTAL SEPTA	MACROVESCULAR STEATOSIS	0.05
19-3423	SK	ND	ND	1	1.2 CM	DISTORTED HEPATIC ARCHITECTURE WITH ATTEMPTED NODULE FORMATION SEPTA BY VARIABLY SIZED SEPTA	AB	AB
19-3430	SK	ND	ND	1	1.2 CM	MAINTAINED HEPATIC ARCHICTECTURE	AB	AB
19-5034	S	ND	ND	1	1.0CM	MAINTAINED	AB	AB
19-5167	MMB	ND	ND	1	1.7 CM	FOCAL DISTORTION WITH FIBROSIS	AB	AB
19-5170	SG	ND	ND	1	1.5 CM	MAINTAINED ARCHICTECTURE	AB	AB
19-5177	BG	ND	ND	3	0.2,0.3 &2.4 CM	DISTORTED ARCHITECTURE WITH ATTEMPTED NODULE FORMATION	MACROVESCULAR STEATOSIS	0.25
19-5199	KK	ND	ND	4	0.1,0.2,0.2,0.5 CM	DILATED PORTAL AND LOBULATED ARCHITECTURE AND NODULE FORMATION	MACROVESCULAR STEATOSIS	0.15
19-8185	KJ	ND	ND	1	1.2 CM	DISTORTED WITH NODULE FORMATION	AB	0.1
19-8229	TS	ND	ND	1	1 CM	LOBULAR DISARRAY	MACROVESCULAR STEATOSIS	0.7
19-8284	BS	ND	ND	2	1 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
19-8320	GG	ND	ND	1	1.3 CM	DISTORTED PORTAL AND LOBULAR ACHITECTURE WITH NODULE FORMATION AND PORTO PORTAL SEPAL	AB	AB
19-8494	MK	ND	ND	4	0.1,0.1,0.1,0.3 CM	DISTORTED PORTAL AND LOBULAR ACHITECTURE WITH NODULE FORMATION	AB	AB
19-8556	S	23	ND	1	0.4 CM	DISTORTED PORTAL AND LOBULAR ACHITECTURE WITH NODULE FORMATION	AB	AB
19-8886	CD	ND	ND	2	1.2, 1.5CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
19-8907	RR	ND	ND	1	1.2 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
19-9089	SS	ND	ND	1	1.6 CM	FOCAL LOBULAR DISARRAY WITH OCCASIONAL THIN PORTO PORTAL SEPTA	MACROVESCULAR STEATOSIS	0.25
19-9097	KD	ND	17.2	1	1.3 CM	DISTORTED PORTAL AND LOBULAR ACHITECTURE WITH NODULE FORMATION SEPRATED BY THCK FIBROUS SEPTA	AB	AB
19-9209	AD	ND	ND	1	1.6 CM	ATTEMPTED NODULAR FORMATION	MACROVESICULAR	0.3
18-9255	PS	ND	ND	1	1.1 CM	MODERATE LOBULAR DISSARY WITH FOCAL ARCHITECTURE DISTORTION	MACRO AND MICRO NUCLEAR	0.6
19-9391	MK	ND	ND	2	0.2 , 10.4 CM	MAINTAINED HEPATIC ARCHICTECTURE	AB	AB
19-9430	JS	ND	ND	1	1.5 CM	FOCAL ARCHITECTURE DISTORTION WITH INCREASED PORTAL FIBROSIS ACUTE PERIPORTAL SEPTA AND SOLITARY PORTO PORTAL SEPTA	MACROVESICULAR	0.1
19-9472	MA	ND	ND	3	0.2,0.4,1.5 CM	MAINTAINED HEPATIC ARCHICTECTURE WITH FOCAL LOBULAR DISARRAY	AB	AB
19-9480	RK	ND	ND	1	1.5 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE SEPRATED BY PORTAL SEPTA	MACROVESICULAR	0.05
19-9503	N	ND	ND	MULTIPLE	0.1 TO 0.2 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE WITH ATTEMPTED NODULE FORMATION SEPTATED BY THEN PORTO PORTAL SEPTATE	MACROVESICULAR	0.05
19-10485	YDS	ND	ND	1	1.2 CM	FOCAL ARCHITECTURE DISTORTION WITH INCREASED P-P SEPLOC	MACROVESICULAR	0.3
19-10535	HS	ND	ND	1	1 CM	DISTORTED WITH NODULE FORMATION	AB	AB
19-10889	KD	ND	ND	2	1.8 AND 1 CM	FOCAL ARCHICTEURE DISTORTION WITH ATTEMPTED NODULE FORMATION	AB	AB

HISTO NO	INITIALS	CHOLESTASIS	BILE	BILE DUCT	MALLORY DENK BODIES
18-9250	HS	AB	AB	AB	OCCASIONAL
18-10276	AK	CANNALICULAR AND INTREACELLULAR TYPE	BILE PLUGS	AB	AB
18-10478	SV	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
18-11287	DK	AB	AB	DUCTULAR PROLIFERATION NOTED	AB
18-11450	KS	INTRACELLULAR BILIRUBINESTASIS AND OCCASIONAL BILEPLUG	BILE DUCT PRILIFERATION	AB	AB
18-11967	RM	AB	AB	AB	AB
18-12298	GS	AB	AB	AB	AB
18-12369	UR	AB	AB	AB	AB
18-12464	JK	AB	AB	AB	AB
18-12563	JB	AB	AB	AB	AB
18-13610	HS	AB	BILE PLUGS	AB	AB
18-13861	PS	AB	BILE PLUGS	AB	AB
18-14352	MM	AB	AB	AB	AB
18-14363	NK	INTRACELLULAR	AB	AB	AB
18-14706	PK	AB	AB	PROLIFERATION	AB
18-14705	M	AB	AB	AB	AB
18-14954	AR	AB	AB	AB	AB
18-14994	SR	AB	AB	AB	AB
18-15205	RS	AB	AB	AB	AB
18-15257	P	AB	AB	AB	BILE DUCT
18-15395	RK	CHOLESTASIS	BILE PLUGS	PROLIFERATION	AB
18-15404	AS	AB	AB	AB	AB
18-15545	AK	P	AB	AB	AB
18-15559	KK	AB	AB	AB	AB
18-15709	SK	AB	AB	AB	AB
18-15734	DK	AB	AB	AB	AB
18-15818	LA	AB	AB	AB	AB
19-52	SG	AB	AB	AB	AB
19-64	N	AB	AB	No DUCTOPENIA	AB
19-190	KK	INTRAHEPATIC	AB	DUCTOPENIA	AB
19-214	JS	AB	AB	DUCTULAR PROLIFERATION	AB
19-323	SS	AB	AB	AB	AB
19-334	AKB	AB	AB	AB	AB
19-385	GS	CANALICULAR	AB	AB	AB
19-639	SK	AB	AB	AB	AB
19-726	RK	AB	AB	AB	AB
19-1099	RS	AB	AB	PROLIFERATION	AB
19-2040	K	AB	AB	AB	AB
19-2047	KS	AB	AB	AB	AB
19-2100	BS	CANALICULAR	BILE PLUGS	PROLIFERATION	AB
19-2086	JK	AB	AB	AB	AB
19-2191	MB	AB	AB	AB	AB
19-2257	TS	AB	AB	PROLIFERATION	AB
19-2316	MK	AB	AB	AB	AB
19-2327	AS	AB	AB	AB	AB
19-2384	BS	AB	BILE PLUGS	AB	AB
19-2774	VA	AB	BILE PLUGS	DUCTOPENIA	AB
19-2813	M	AB	AB	AB	AB
19-2904	VA	P	AB	PROLIFERATION	AB
19-2923	PS	AB	AB	AB	AB
19-3012	SM	CANALICULAR	P	AB	AB
19-3423	SK	AB	AB	BILE DUCT PROLIFERATION	
19-3430	SK	AB	AB	AB	AB
19-5034	S	AB	AB	AB	AB
19-5167	MMB	AB	AB	P	AB
19-5170	SG	AB	AB	AB	AB
19-5177	BG	AB	AB	AB	AB
19-5199	KK	AB	AB	AB	AB
19-8185	KJ	AB	AB	AB	AB
19-8229	TS	AB	AB	AB	AB
19-8284	BS	AB	AB	AB	AB
19-8320	GG	P	AB	P	AB
19-8494	MK	CANALICULAR	BILE PLUGS	AB	AB
19-8556	S	AB	AB	AB	AB
19-8886	CD	AB	AB	AB	AB
19-8907	RR	AB	AB	AB	AB
19-9089	SS	AB	AB	ABSENT	AB
19-9097	KD	AB	AB	ABSENT	AB
19-9209	AD	AB	AB	ABSENT	AB
18-9255	PS	AB	AB	ABSENT	AB
19-9391	MK	AB	AB	ABSENT	AB
19-9430	JS	AB	AB	ABSENT	AB
19-9472	MA	BILE PLUG	AB	DUCTAL PROLIFERATION NOTED	AB
19-9480	RK	MILD INTRACELLULAR CHOLESTASIS	AB	ABSENT	AB
19-9503	N	AB	AB	ABSENT	AB
19-10485	YDS	AB	AB	DUCTAL PROLIFERATION NOTED	P
19-10535	HS	P	AB	ABSENT	AB
19-10889	KD	AB	AB	ABSENT	AB

HISTO NO	INITIALS	HEPATOCYTES
18-9250	HS	MILD BALLOONING DEGENRATION
18-10276	AK	BALLOONING DEGENERATION
18-10478	SV	MILD BALLOONING DEGENERATION
18-11287	DK	MILD BALLOONING DEGENERATION
18-11450	KS	WELL FORMED EPITHELIOD GRANULOMAS
18-11967	RM	PRESENCE OF FINE GRANULAR BROWN PIGMENT
18-12298	GS	NP
18-12369	UR	NP
18-12464	JK	MILD BALLOONING DEGENRATION
18-12563	JB	PRESENCE OF EPITHELIOID CELL GRANULOMA ALONG WITH LANGHAN'S AND FOREIGN BODY GIANT CELLS SURROUNDED BY LARGE AREAS OF FIBROSIS
18-13610	HS	ACIDOPHILIC BODIES SEEN
18-13861	PS	FINE TO COARSE INTRACELLULAR BROWN PIGMENT
18-14352	MM	BALLOONING DEGENERATION
18-14363	NK	MILD BALLOONING DEGENERATION
18-14706	PK	MILD BALLOONING DEGENERATION
18-14705	M	MILD BALLOONING DEGENERATION
18-14954	AR	EXTRACELLULAR DEPOSITION OF PINK MATERIAL
18-14994	SR	MILD BALLOONING DEGENERATION
18-15205	RS	MILD BALLOONING DEGENERATION
18-15257	P	MODERATE BALLOONING DEGENRATION
18-15395	RK	MILD BALLOONING DEGENERATION
18-15404	AS	EPITHELIOD CELL GRANULOMAS
18-15545	AK	BALLOONING DEGENERATION
18-15559	KK	MILD BALLOONING DEGENERATION
18-15709	SK	GRANULOMAS
18-15734	DK	UR
18-15818	LA	NP
19-52	SG	MILD BALLOONING DEGENERATION
19-64	N	MILD BALLOONING DEGENERATION
19-190	KK	MILD BALLOONING DEGENERATION
19-214	JS	MODERATE BALLOONING DEGENRATION,
19-323	SS	MODERATE BALLOONING DEGENRATION
19-334	AKB	NP
19-385	GS	MILD BALLOONING DEGENERATION
19-639	SK	MILD BALLOONING DEGENERATION
19-726	RK	NP
19-1099	RS	MILD BALLOONING DEGENERATION
19-2040	K	MILD BALLOONING DEGENERATION
19-2047	KS	MILD BALLOONING DEGENERATION
19-2100	BS	BALLOONING DEGENERATION
19-2086	JK	MILD BALLOONING DEGENERATION
19-2191	MB	MILD BALLOONING DEGENERATION
19-2257	TS	MILD BALLOONING DEGENERATION
19-2316	MK	EMPERIPOLESIS AND HEPATOCYTIC ROSSETTING
19-2327	AS	NP
19-2384	BS	MILD BALLOONING DEGENERATION
19-2774	VA	MILD BALLOONING DEGENERATION
19-2813	M	UR
19-2904	VA	MILD BALLOONING DEGENERATION
19-2923	PS	MODERATE BALLOONING DEGENRATION
19-3012	SM	MILD BALLONING DEGENRATION
19-3423	SK	MILD BALLOONING DEGENERATION
19-3430	SK	UR
19-5034	S	EXTRACELLULAR DEPOSITION OF PINK MATERIAL
19-5167	MMB	MILD BALLOONING DEGENERATION
19-5170	SG	UR
19-5177	BG	MILD BALLOONING DEGENERATION
19-5199	KK	MILD BALLOONING DEGENERATION
19-8185	KJ	MILD BALLOONING DEGENERATION
19-8229	TS	MILD BALLOONING DEGENERATION
19-8284	BS	NP
19-8320	GG	MILD BALLOONING DEGENERATION
19-8494	MK	MILD BALLOONING DEGENERATION
19-8556	S	MILD BALLOONING DEGENERATION
19-8886	CD	MILD BALLOONING DEGENERATION
19-8907	RR	MILD BALLOONING DEGENERATION
19-9089	SS	MILD BALLOONING DEGENERATION
19-9097	KD	AMYLOID DEPOSITS
19-9209	AD	MODERATE BALLOONING DEGENRATION
18-9255	PS	MILD BALLOONING DEGENRATION
19-9391	MK	UR
19-9430	JS	NP
19-9472	MA	MILD BALLOONING DEGENERATION
19-9480	RK	MILD BALLOONING DEGENERATION
19-9503	N	MILD BALLOONING DEGENERATION
19-10485	YDS	MILD BALLOONING DEGENERATION
19-10535	HS	MILD BALLOONING DEGENERATION
19-10889	KD	MILD BALLOONING DEGENERATION

HISTO NO	INITIALS	INFLAMMATORY INFILTRATE PORTAL TRACTS/SEPTA/SINUSOIDS	PLASMA CELL RICH	GRANULOMA	INTERFACE HEPATITIS	SPOTTY HEPATIC NECROSIS
18-9250	HS	ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	AB	P	AB
18-10276	AK	DILATION INFILTRATED BYY MILD TO MODERAT DEGREE OF INFILTRATE LYMPHOCYTES, HISTIOCYTES, OCCASIONAL PLASMA CELLS AND EOSINOPHILS	AB	AB	P	AB
18-10478	SV	MODERATE DEGREE OF LYMPHOMONONUCLEAR CELL INFILTRATE MAINLY LYMPHOCYTES	AB	AB	P	AB
18-11287	DK	MILD LYMPHONUCLEAR CELLS INFILTRATED WITH LYMPHOCYTES, HISTIOCYTES AND FEW PLASMA CELLS	AB	AB	P	AB
18-11450	KS	UNREMARKABLE	AB	AB	AB	AB
18-11967	RM	UNREMARKABLE	AB	AB	AB	AB
18-12298	GS	MODERATE DEGREE OF INFILTRATING INFILTRATE COMPRISING OFLYMPHOCYTES, HISTIOCYTES ALONG WITH PRESENCE OF FEW LYMPHOID AGGREGATES AND INFILTRATION WITH INCREASED FIBROSIS	AB	AB	P	AB
18-12369	UR	MINIMAL INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES.	AB	AB	AB	AB
18-12464	JK	DENSE INFLAMMATORY INFILTRATE COMPRISING OF LYMPHOCYTES AND PLASMA CELLS	AB	AB	P	AB
18-12563	JB	INFLAMMATORY INFILTRATE LYMPHOCYTES AND HISTIOCYTES.	AB	AB	P	AB
18-13610	HS	INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, PLASMA CELLS, EOSINOPHILS AND OCCASIONAL HISTIOCYTES	AB	AB	AB	AB
18-13861	PS	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATES	AB	AB	AB	AB
18-14352	MM	MODERATE INFILTRATION BY INFLAMMATORY INFILTRATE MAILY NEUTROPHILS, LYMPHOCYTES AND EOSINOPHILS	AB	AB	P	P
18-14363	NK	MILD INFILTRATION BY LYMPHOCYTES, HISTIOCYTES AND EOSINOPHILS	AB	AB	P	AB
18-14706	PK	DENSE INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHICYTES, HISTIOCYTES AND FEW PLASMA CELLS	AB	AB	P	AB
18-14705	M	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
18-14954	AR	PINK ACELLULAR MATERIAL	AB	AB	AB	AB
18-14994	SR	INFILTRATION BY LYMPHOCYTES, HISTIOCYTES AND PLASMA CELLS	AB	AB	P	AB
18-15205	RS	MODERATE INFILTRATION BY LYMPHOCYTES	AB	AB	P	AB
18-15257	P	MILD INFILTRATION BY MODERATE CHRONIC INFLAMMATORY INFILTRATE CELLS SEEN AS LYMPHOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
18-15395	RK	MODERATE DEGREE OF MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES, HISTIOCYTES AND EOSINOPHILS	AB	AB	P	AB
18-15404	AS	MILD TO MODERATE DEGREE OF MIXED INFLAMMATORY INFILTRATE WITH SMALL EPITHELIOID CELL GRANULOMAS NOTED.	AB	AB	AB	AB
18-15545	AK	MODERATE INFILTRATION BY INFLAMMATORY INFILTRATE MAINLY LYMPHOCYTES, PLASMA CELLS, EOSINOPHILS AND HISTIOCYTES	AB	AB	P	AB
18-15559	KK	DENSE INFILTRATION BY INFLAMMATORY INFILTRATE MAINLY LYMPHOCYTES, HISTIOCYTES AND FEW EOSINOPHILS	AB	AB	MILD	AB
18-15709	SK	UNREMARKABLE	AB	P	AB	AB
18-15734	DK	MINIMAL INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND FEW EOSINOPHILS	AB	AB	AB	AB
18-15818	LA	MODERATE INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND OCCASIONAL LYMPHOID AGGREGATE	AB	AB	AB	AB
19-52	SG	MILD FIBROSIS WITH DENSE DEGREE OF INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	FOCAL	P
19-64	N	MILD INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND OCCASIONAL PLASMA CELLS	AB	AB	AB	AB
19-190	KK	DENSE INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES	AB	AB	AB	AB
19-214	JS	DENSE INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHICYTES AND HISTIOCYTES	AB	AB	P	AB
19-323	SS	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
19-334	AKB	MODERATE INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES.	AB	AB	P	AB
19-385	GS	NO SIGNIFICANT INFLAMMATORY INFILTRATE SEEN.	AB	AB	AB	P
19-639	SK	DENSE INFILTRATION BY LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
19-726	RK	MILD INFILTRATION OF PORTAL TRACT BY CHRONIC INFLAMMATORY CELLS	AB	AB	AB	AB
19-1099	RS	INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES, OCCASIONAL NEUTROPHILS AND PLASMA CELLS	AB	AB	P	P
19-2040	K	MILD INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES	AB	AB	AB	AB
19-2047	KS	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
19-2100	BS	DENSE INFILTRATION BY LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
19-2086	JK	MIODERATE DEGREE OF INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND PLASMA CELLS	AB	AB	P	AB
19-2191	MB	MILD INFLAMMATORY INFILTRATE	AB	AB	AB	AB
19-2257	TS	MODERATE INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES, FEW PLASMA CELLS AND NEUTROPHILS	AB	AB	FOCAL	AB
19-2316	MK	DENSE INFILTRATION BY LYMPHOCYTES, HISTIOCYTES, EOSINOPHILS AND FEW PLASMA CELLS	AB	AB	P	AB
19-2327	AS	MILD INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES	AB	AB	AB	AB
19-2384	BS	PORTAL FIBROSIS WITH PORTO PORTAL SEPTAL. DENSE INFILTRATION BY LYMPHOCYTES AND PLASMA CELLS	AB	AB	AB	AB
19-2774	VA	INCREASED FIBROSIS AND INFILTRATION BY MODERATE MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS AND LYMPHOCYTES	AB	AB	AB	AB
19-2813	M	MINIMAL INFILTRATION BY LYMPHOCYTES	AB	AB	AB	AB
19-2904	VA	ENLARGED WITH FIBROSIS	AB	AB	AB	AB
19-2923	PS	MODERATE INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
19-3012	SM	MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTARTE INFLAMMATORY INFILTRATE WITH OCCASIONAL PORTO PORTAL SEPTAL	AB	AB	AB	AB
19-3423	SK	MODERATE INFILTRATION BY LYMPHOCYTES AND FEW PLASMA CELLS	AB	AB	P	AB
19-3430	SK	SHOW MILD INFILTRATION BY LYMPHOCYTES AND PLASMA CELLS. FOCI INCREASE IN PORTAL FIBROSIS SEEN	AB	AB	AB	AB
19-5034	S	EOSINOPHILIC ACELLULAR GLOBULAR STRUCTURES	AB	AB	AB	AB
19-5167	MMB	MODERAQTE INFILTRATION BY LYMPHOCYTES, PLASMA CELLS AND FEW HISTIOCYTES	AB	AB	AB	AB
19-5170	SG	OCCASIONAL LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
19-5177	BG	DENSE CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, PLASMA CELLS AND HISTIOCYTES	AB	AB	P	AB
19-5199	KK	MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND OCCASIONAL PLASMA CELLS.	AB	AB	AB	AB
19-8185	KJ	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
19-8229	TS	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES.	AB	AB	AB	AB
19-8284	BS	MILD TO MODERATE INFILTRATION SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	P
19-8320	GG	DENSE INFILTRATION BY LYMPHOCYTES AND PLASMA CELLS, HISTIOCYTES AND FEW EOSINOPHILS	AB	AB	P	AB
19-8494	MK	MILD TO MODERATE INFILTRATION BY LYMPHOCYTES AND HISTIOCYTES	AB	AB	FOCAL SPILLOVERS	AB
19-8556	S	MODERATE INFILTRATION BY INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES AND HISTIOCYTES.	AB	AB	AB	AB
19-8886	CD	MIXED INFLAMMATORY INFILTRATE SEEN AS LMPHOCYTES , HISTIOCYTES, FEW NEUTROPHILSAND PLASMA CELLS	AB	AB	FOCAL SPILLOVERS	AB
19-8907	RR	MILD TO MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND FEW NEUTROPHILS	AB	AB	AB	AB
19-9089	SS	MILD TO MODERATE MUCOID INFLAMMATORY INFILTRATE BY LYMPHOCYTES AND HISTIOCYTES. OCCASIONAL LOBULAR INFLAMMATION	AB	AB	AB	AB
19-9097	KD	MODERATE INFILTRATION BY NO INFLAMMATORY SEEN AS LYMPHOCYTES AND HISTIOCYTES. AMYLOID DEPOSITS SEEN.	AB	AB	AB	AB
19-9209	AD	MILD CHRONIC INFLAMMATORY INFILTRATE	AB	AB	AB	AB
18-9255	PS	FIBROSIS AND MIXED INFLAMMATORY INFILTRATE WITH PERIPORTAL,PERVENULAR AND PORTAL FIBROSIS AND OCCASIONAL PORTOPORTAL SEPTA	AB	AB	AB	AB
19-9391	MK	NO	AB	AB	AB	AB
19-9430	JS	MODERATE LYMPHOCYTIC INFILTRATION	AB	AB	AB	P
19-9472	MA	MODERATE INFILTRATION BY MOXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, EOSINOPHILS AND LYMPHOCYTES	AB	AB	AB	AB
19-9480	RK	MODERATE TO MARKED INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES, NEUTROPHILS AND EOSINOPHILS	AB	AB	AB	AB
19-9503	N	CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND PLASMA CELLS	AB	AB	MODERATE	AB
19-10485	YDS	MODERATE INFLAMMATORY INFILTRATE WITH LYMHOCYTES AND HISTIOCYTES	AB	AB	FOCAL SPILLOVERS	AB
19-10535	HS	MODERATE MIXED INFLAMMATORY INFILTRATE	AB	AB	P	AB
19-10889	KD	MODERATE TO DENSE INFILTRATION	AB	AB	FOCAL	P

HISTO NO	INITIALS	CONFLUENT NECROSIS	FIBROSIS	STAINS		
				IRON	MASSON TRICHOME	RETICULIN
18-9250	HS	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTAL SCARS WITH PERICELLULAR PERIVENULAR FIBROSIS ALONG WITH ATTEMPTED NODULE FORMATION	MAINTAINED
18-10276	AK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-10478	SV	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULAR FORMATION WITH FOCAL PERICELLULAR FIBROSIS	MAINTAINED
18-11287	DK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-11450	KS	AB	AB	ND	ND	ND
18-11967	RM	AB	AB	NEGATIVE	DOES NOT HIGHLIGHTS ANY INCREASE FIBROSIS.	MAINTAINED
18-12298	GS	AB	GARDE II	NEGATIVE	INCREASED PORTAL FIBROSIS AND OCCASIONAL THIN SEPTAL	MAINTAINED
18-12369	UR	AB	AB	NEGATIVE	DOES NOT HIGHLIGHT ANY INCREASED PORTAL FIBROSIS	MAINTAINED
18-12464	JK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-12563	JB	AB	AB	ND	ND	EXTENSIVELY HIGHLIGHTS GRANULOMAS
18-13610	HS	P	AB	NEGATIVE	DOES NOT HIGHLIGHTS INCREASED FIBROSIS	NORMAL
18-13861	PS	AB	AB	NEGATIVE	DOES NOT HIGHLIGHT ANY INCREASED FIBROSIS	MAINTAINED
18-14352	MM	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-14363	NK	AB	AB	NEGATIVE	DOES NOT HIGHLIGHT ANY INCREASED FIBROSIS	MAINTAINED
18-14706	PK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-14705	M	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
18-14954	AR	AB	AB	ND	ND	ND
18-14994	SR	AB	GRADE IV	NEGATIVE	NODULE FORMATION	MAINTAINED
18-15205	RS	AB	GRADE IV	NEGATIVE	HIGHLIGHT NODULE FORMATION	MAINTAINED
18-15257	P	AB	GRADE IV	NEGATIVE	NODULE FORMATION	MAINTAINED
18-15395	RK	AB	GRADE II	NEGATIVE	INCREASED PORTAL FIBROSIS	MAINTAINED
18-15404	AS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
18-15545	AK	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS	MAINTAINED
18-15559	KK	AB	GRADE IV	NEGATIVE	PORTAL,PERIPORTAL AND PERICELLULAR FIBROSIS	MAINTAINED
18-15709	SK	AB	AB	ND	ND	ND
18-15734	DK	AB	AB	NEGATIVE	NO INCREASED FIBROSIS	MAINTAINED
18-15818	LA	AB	AB	NEGATIVE	NO INCREASED FIBROSIS	MAINTAINED
19-52	SG	P	GRADE II	NEGATIVE	MILD INCREASE IN FIBROSIS	MAINTAINED
19-64	N	AB	GRADE III	NEGATIVE	HIGHHLIGHTS MICRONODULE FORMATION AT PLACES	MAINTAINED
19-190	KK	AB	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-214	JS	AB	GRADE IV	POSITIVITY IN FEW KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-323	SS	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED FIBROSIS WITH PORTO PORTAL AND PORTOVESCULAR SEPTAL	MAINTAINED
19-334	AKB	AB	GRADE IV	NEGATIVE	HIGHLIGHT NODULE FORMATION	MAINTAINED
19-385	GS	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS INCREASED FIBROSIS	MAINTAINED
19-639	SK	AB	GRADE III	NEGATIVE	HIGHLIGHTS PORTO-PORTAL SEPTAL WITH ATTEMPTED NODULE FORMATION	MAINTAINED
19-726	RK	AB	AB	NEGATIVE	PORTAL FIBROSIS IN AN OCCASIONAL PORTAL TRACTS SHOWS MAINTAINED HEPATIC FRAMEWORK	MAINTAINED
19-1099	RS	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
19-2040	K	AB	AB	NEGATIVE	NO INCREASED FIBROSIS	MAINTAINED
19-2047	KS	AB	AB	NEGATIVE	NO INCREASED FIBROSIS	MAINTAINED
19-2100	BS	AB	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS	FOCAL COLLAPSE
19-2086	JK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-2191	MB	AB	GRADE I	NEGATIVE	HIGHLIGHTS MILD INCREASED IN PORTAL FIBROSIS	MAINTAINED
19-2257	TS	AB	GRADE III	NEGATIVE	ATTEMPTED NODULE FORMATION	MAINTAINED
19-2316	MK	AB	GRADE III	NEGATIVE	ATTEMPTED NODULE FORMATION	MAINTAINED
19-2327	AS	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
19-2384	BS	AB	GRADE III	NEGATIVE	HIGHLIGHTS FIBROSIS	MAINTAINED
19-2774	VA	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS	MAINTAINED
19-2813	M	AB	GRADE II	NEGATIVE	INCREASED PORTAL FIBROSIS	MAINTAINED
19-2904	VA	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL AND SEPTAL FIBROUS WITH OCCASIONAL FOCUS OF MILD PERIVENDULAR	MAINTAINED
19-2923	PS	AB	GRADE II	NEGATIVE	PORTAL FIBROSIS WILL PORTO PORTAL SEPTAL AND OCCASIONAL ATTEMPTED NODULE FORMATION	NORMAL
19-3012	SM	AB	GRADE II	NEGATIVE	HIGHLIGHTS PORTO PORTAL SEPTAL	MAINTAINED
19-3423	SK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION WITH PERICELLULAR FIBROSIS	MAINTAINED
19-3430	SK	AB	GRADE III	NEGATIVE	HGIGHLIGHTS FOCAL INCREASE IN PORTAL	MAINTAINED
19-5034	S	AB	AB	AB	AB	ND
19-5167	MMB	AB	GRADE IV	NEGATIVE	HIGHLIGHT NODULE FORMATION	MAINTAINED
19-5170	SG	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
19-5177	BG	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-5199	KK	AB	GRADE IV	NEGATIVE	HIGHTLIGHTS NODULE FORMATION	MAINTAINED
19-8185	KJ	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-8229	TS	AB	GRADE II	NEGATIVE	HIGHLIGHTS INCREASED PORTAL, PERI PORTAL, PERICELLULAR FIBROSIS AND OCCASIONAL PORTO PORTAL SEPTAL	MAINTAINED
19-8284	BS	AB	GRADE III	POSITIVITY IN FEW KUPFFER CELLS	HIGHLIGHTS INCREASED FIBROSIS IN PORTAL PORT	MAINTAINED
19-8320	GG	AB	GRADE II	NEGATIVE	INCREASED PORTAL FIBROSIS IN PORTAL WITH COMPLETE NODULE FORMATION	MAINTAINED
19-8494	MK	AB	GRADE IV	POSITIVITY IN FEW KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-8556	S	AB	GRADE IV	NEGATIVE	NODULE FORMATION AND FOCAL PERI CELLULAR FIBROSIS	MAINTAINED
19-8886	CD	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-8907	RR	AB	AB	NEGATIVE	HIGHLIGHTS MILD PORTAL FIBROSIS	MAINTAINED
19-9089	SS	AB	2PERICELLULAR	NEGATIVE	HIGHLIGHTS THIN PORTO PORTAL SEPTAL.	MAINTAINED
19-9097	KD	AB	GRADE IV	POSITIVITY IN KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-9209	AD	AB	GRADE IV	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
18-9255	PS	AB	GRADE III	FOCAL POSITIVITY IN KUPFFER CELLS	HIGHLIGHTS PERICELLULAR, PERIVENULAR AND PORTAL FOCALLY FIBROSIS WITH OCCASIONAL SEPTA	COLLAPSE
19-9391	MK	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS FIBROSIS	MAINTAINED
19-9430	JS	AB	GRADE III	NEGATIVE	HIGHLIGHTS INCREASED PORTAL FIBROSIS AND PERIPORTAL SEPTAL	MAINTAINED
19-9472	MA	AB	GRADE I	FOCALLY POSITIVE IN KUPFFER CELLS	HIGHLIGHTS FOCAL PERICELLLAR FIBROSIS	MAINTAINED
19-9480	RK	AB	GRADE IV	FOCAL POSITIVITY IN KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-9503	N	P	GRADE III	NEGATIVE	ATTEMPTED NODULE FORMATION	COLLAPSE
19-10485	YDS	AB	GRADE III	MODERATE PIGMENT DEPOSITION	PORTO PORTAL SEPTA	MAINTAINED
19-10535	HS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-10889	KD	AB	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED

HISTO NO	INITIALS	OTHERS
18-9250	HS	ND
18-10276	AK	ND
18-10478	SV	ND
18-11287	DK	ND
18-11450	KS	20% ZN AFB IS NEGATIVE
18-11967	RM	ND
18-12298	GS	ND
18-12369	UR	ND
18-12464	JK	ND
18-12563	JB	20% ZN AFB IS NEGATIVE
18-13610	HS	ND
18-13861	PS	ND
18-14352	MM	ND
18-14363	NK	ND
18-14706	PK	ND
18-14705	M	ND
18-14954	AR	CONGO RED STAIN REVEALS PINK DEPOSITS AND APPLE GREEN BIREFRINGENCE ON POLARISED LIGHT.
18-14994	SR	ND
18-15205	RS	ND
18-15257	P	ND
18-15395	RK	ND
18-15404	AS	20% ZN- NEGATIVE
18-15545	AK	ND
18-15559	KK	ND
18-15709	SK	20% ZN- NEGATIVE
18-15734	DK	ND
18-15818	LA	ND
19-52	SG	ND
19-64	N	ORECIN AND RHODODENDRON FOR COPPER SHOW MILD FOCAL PRESENCE OF COPPER IN PERISEPTAL REGION
19-190	KK	ND
19-214	JS	ND
19-323	SS	ND
19-334	AKB	ND
19-385	GS	ND
19-639	SK	ND
19-726	RK	ND
19-1099	RS	ND
19-2040	K	ND
19-2047	KS	ND
19-2100	BS	ND
19-2086	JK	ND
19-2191	MB	ND
19-2257	TS	ND
19-2316	MK	ND
19-2327	AS	ND
19-2384	BS	ND
19-2774	VA	ND
19-2813	M	ND
19-2904	VA	ND
19-2923	PS	NEGATIVE
19-3012	SM	ND
19-3423	SK	ND
19-3430	SK	HEPATIC PARENCHYMA
19-5034	S	CONGO RED POSITIVE FOR AMYLOID ON POLARISED LIGHT SHOWING APPLE GREEN BIREFRINGENCE
19-5167	MMB	ND
19-5170	SG	ND
19-5177	BG	ND
19-5199	KK	ND
19-8185	KJ	ND
19-8229	TS	ND
19-8284	BS	ND
19-8320	GG	ND
19-8494	MK	ND
19-8556	S	ND
19-8886	CD	ND
19-8907	RR	ND
19-9089	SS	ND
19-9097	KD	CONGO RED POSITIVE FOR AMYLOID ON POLARISED LIGHT SHOWING APPLE GREEN BIREFRINGENCE
19-9209	AD	ND
18-9255	PS	ND
19-9391	MK	ND
19-9430	JS	ND
19-9472	MA	ND
19-9480	RK	ND
19-9503	N	ND
19-10485	YDS	ND
19-10535	HS	ND
19-10889	KD	ND

HISTO NO	INITIALS	IMPRESSION
18-9250	HS	ASH GRADE 6 STAGE 3
18-10276	AK	NASH GRADE 6 STAGE 4
18-10478	SV	NASH GRADE 4 STAGE 4
18-11287	DK	NASH GRADE 4 STAGE 4
18-11450	KS	GRANULOMATOUS HEPATITIS- TUBERCULOSIS
18-11967	RM	NO SPECIFIC PATHOLOGY- R/O GILBERT SYNDROME
18-12298	GS	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MILD ACTIVITY (HAI SCORE 5, GRADE 2, STAGE 2)
18-12369	UR	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MINIMAL ACTIVITY, HAI SCORE 1, GRADE 1 , STAGE 0
18-12464	JK	AUTOIMMUNE HEPATITIS
18-12563	JB	GRANULOMATOUS INFLAMMATION WITH SCLEROSIS- SARCOIDOSIS
18-13610	HS	ACUTE HEPATITIS (HAV POSITIVE)
18-13861	PS	CHOLESTATIC LIVER DISEASE- R/O DUBIN JOHNSON SYNDROME
18-14352	MM	AUTOIMMUNE HEPATITIS
18-14363	NK	ACUTE HEPATITIS (HAV POSITIVE)
18-14706	PK	AUTOIMMUNE HEPATITIS
18-14705	M	NASH GRADE 3 STAGE 4
18-14954	AR	AMYLOIDOSIS
18-14994	SR	AUTOIMMUNE HEPATITIS
18-15205	RS	PRIMARY BILIARY CIRRHOSIS
18-15257	P	NASH GRADE 3 STAGE 4
18-15395	RK	LARGE DUCT OBSTRUCTION
18-15404	AS	GRANULOMATOUS HEPATITIS- TUBERCULOSIS
18-15545	AK	AUTOIMMUNE HEPATITIS
18-15559	KK	NASH GRADE 6 STAGE 4
18-15709	SK	SARCOIDOSIS
18-15734	DK	NO SPECIFIC PATHOLOGY
18-15818	LA	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1, STAGE 0
19-52	SG	CHRONIC HEPATITIS- IDIOPATHIC
19-64	N	AUTOIMMUNE HEPATITIS
19-190	KK	NASH GRADE 4 SATGE 4
19-214	JS	ASH GRADE 3 STAGE 4
19-323	SS	NASH GRADE 5 STAGE 3
19-334	AKB	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 0, GRADE ,1 STAGE 1
19-385	GS	DILI(HERBAL MEDICINES)
19-639	SK	NASH GRADE 4 STAGE 3
19-726	RK	CHRONIC VIRAL HEPATITIS(HCV POSITIVE) HAI SCORE 3, GRADE 2,STAGE 1
19-1099	RS	CHRONIC HEPATITIS- IDIOPATHIC
19-2040	K	NASH GRADE 4 STAGE 0
19-2047	KS	NASH GRADE 4 STAGE 0
19-2100	BS	ACUTE HEPATITIS (HAV)
19-2086	JK	AUTOIMMUNE HEPATITIS
19-2191	MB	AUTOIMMUNE HEPATITIS
19-2257	TS	NASH GRADE 4 STAGE 4
19-2316	MK	AUTOIMMUNE HEPATITIS
19-2327	AS	CHRONIC VIRAL HEPATITIS (HCV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 2, GRADE 1, STAGE II
19-2384	BS	AUTOIMMUNE HEPATITIS
19-2774	VA	PRIMARY SCLEROSING CHOLANGITIS
19-2813	M	NO SPECIFIC PATHOLOGY
19-2904	VA	LARGE DUCT OBSTRUCTION
19-2923	PS	NASH GRADE 6 STAGE 3
19-3012	SM	CHOLESTATIC LIVER DISEASE- R/O BENIGN RECURRENT INTRAHEPATIC CHOLESTASIS/PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS
19-3423	SK	NASH GRADE 3 STAGE 4
19-3430	SK	NO SPECIFIC PATHOLOGY
19-5034	S	AMYLOIDOSIS
19-5167	MMB	NASH GRADE 3 SATGE 4
19-5170	SG	NO SPECIFIC PATHOLOGY
19-5177	BG	AUTOIMMUNE HEPATITIS
19-5199	KK	NASH GRADE 4 STAGE 4
19-8185	KJ	NASH GARDE 4 STAGE 4
19-8229	TS	NASH GRADE 6 STAGE 2
19-8284	BS	CHRONIC VIRAL HEPATITIS(HCV POSITIVE) WITH MINIMAL ACTIVITY HAI SCORE 3, GRADE 1, STAGE 1
19-8320	GG	CIRRHOSIS- AUTOIMMUNE WITH PRIMARY BILIARY CIRRHOSIS- OVERLAP SYNDROME
19-8494	MK	LARGE DUCT OBSTRUCTION
19-8556	S	NASH GRADE 4 STAGE 4
19-8886	CD	AUTOIMMUNE HEPATITIS
19-8907	RR	ACUTE HEPATITIS (HAV POSITIVE)
19-9089	SS	NASH GRADE 5, STAGE 2
19-9097	KD	AMYLOIDOSIS
19-9209	AD	NASH GRADE 5 SATGE 4
18-9255	PS	ASH GRADE 4 STAGE
19-9391	MK	NO SPECIFIC PATHOLOGY
19-9430	JS	CHRONIC VIRAL HEPATITIS(HBV POSITIVE) HAI SCORE 2, GRADE 1 , STAGE 2
19-9472	MA	DILI
19-9480	RK	LARGE DUCT OBSTRUCTION
19-9503	N	AUTOIMMUNE HEPATITIS
19-10485	YDS	NASH GRADE 6 STAGE 3
19-10535	HS	LARGE DUCT OBSTRUCTION
19-10889	KD	AUTOIMMUNE HEPATITIS

				HISTORY									BIOCHEMISTRY								
HISTO NO	INITIALS	MRD	AGE/SEX	JAUNDICE	DIABETES	ALCOHAL	OBESITY	ABDOMINAL MASS	HEPATO MEGALY	SPLENOME GALY	LYMPHADENO PATHY	RADIOLOGICAL FINDINGS	BILI TOTAL	BILI DIRECT	SGOT	SGPT	ALBUMIN	ALP	PROTIENS	VIRAL MARKERS	AUTOIMMUNE MARKERS
19-11058	NM	799490	60/F	P	AB	AB	AB	AB	AB	P	AB	MODERATE ASCITIS ,MILD SPLENOMEGALY	14.9	13.18	95	76	2.6	101	6.4	NR	NEGATIVE
19-11278	SK	806988	19/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS LIVER	0.6	0.82	120	140	2.5	126	6.8	NR	ASMA 2+, ANA-2+, SLA-1+ N
19-11395	DD	804150	56/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	0.56	0.13	26	16	2.15	60	5.1	NR	ND
19-11647	AKG	811260	62/M	P	AB	AB	AB	AB	AB	P	AB	ND	4.16	1.52	73	30	1.8	262	8.2	NR	ND
19-12245	SS	822682	57/M	P	AB	AB	AB	AB	P	P	AB	HEPATOSPLENOMEGALY	2	1.6	55	30	3.4	362	7.1	NR	ND
19-12257	JR	332267	62/F	AB	AB	AB	AB	AB	AB	P	AB	MDCT	0.61	0.49	60	26	2.3	347	5.7	NR	ND
19-12612	NS	823062	34/M	P	AB	AB	AB	AB	AB	AB	AB	ND	32.4	24.5	96	26	2.5	159	3.5	NR	AMA 2+, ANA 3+
19-12657	AT	820945	28/M	P	AB	AB	AB	AB	P	P	AB	CLD WITH PORTAL HTN	3.62	2.99	174	75	5.3	161	7.5	NR	ANA +
19-12896	AP	827589	44/M	P	AB	AB	AB	AB	P	P	AB	CLD WITH PORTAL HTN	6.7	5.18	219	105	2.6	169	5.7	NR	ND
19-13088	S	833495	26/M	P	AB	AB	AB	AB	AB	AB	AB	ND	3.8	3.49	192	325	3.9	155	6.9	HEV POSITIVE	ND
19-13523	A	784530	27/M	P	AB	AB	AB	AB	AB	AB	AB	NAD	1.51	0.19	40	42	5.6	121	8.2	NR	ND
19-13625	M	842011	26/F	P	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	6.65	5.29	68	50		549		NR	ANA +VE
19-14106	JS	838674	22/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	4.81	2.69	78	73	5.2	192	6.4	NR	ANA 3+
19-14425	MK	358932	32/F	P	P	AB	AB	AB	P	P	AB	HEPATOSPLENOMEGALY WITH PORTAL HTN	2.1	0.51	85	57	2.2	747	6.2	NR	ND
'19-14560	BG	861694	49/F	P	AB	AB	AB	AB	AB	AB	AB	ASCITIS	1.56	1	49	28	2	253	5.6	NR	AMA 3+
19-14837	SM	855966	52/M	P	AB	AB	AB	AB	AB	P	AB	SPLENOMEGALY	26	19.9	44	66	3.45	149	5.4	NR	NEGATIVE
19-15101	AK	852042	65/F	AB	AB	AB	AB	AB	AB	AB	P	CHRONIC LIVER DISEASE	3.6	3.14	153	92	3.2		8.5	NR	ANA 4+
19-15205	BS	867964	49/M	AB	AB	P	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	11.1	8.48	112	43	2.45	116	7.1	NR	ND
19-15254	CS	868538	62/M	AB	AB	AB	AB	AB	AB	AB	AB	ASCITIS GRADE II	1.8	0.94	51	27	2.8	104	5.9	NR	ND
19-15470	R	869735	6/F	P	AB	AB	AB	AB	AB	AB	AB	MILD HEPATOMEGALY	38.4	28.36	129	73	3.7	303	6.4	NR	ND
19-15479	PS	865619	40/M	AB	AB	AB	AB	AB	AB	AB	AB	MULTIPLE HYPERINTENSE LESIONS IN LIVER?INFECTIVE ? FUNGAL	0.37	0.05	29	10	2.12	97	4.1	NR	ND
19-15531	SS	871284	50/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	0.54	0.14	42	57	2.5	96	5.2	NR	ND
19-15357	VD	866006	65/F	P	AB	AB	AB	AB	P	P	AB	MILD HEPATOMEGALY AND SPLEENOMEGALY	9.96	11.1	131	102	4.1	152	3.6	NR	ND
19-15598	HS	854153	40/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.58	0.72	63	61	3.8	56	5.5	NR	ANA 1+
20-67	A	873699	38/M	P	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	1.29	0.5	41	40	3.5	100	6.4	NR	ND
20-71	RCS	873239	38/M	P	AB	AB	AB	AB	AB	P	AB	ASCITIS WITH SPLEENOMEGALY	5.4	4.8	1207	915	4.2	133	5.6	NR	ASMA 2+
20-353	VS	585645	52/M	P	AB	AB	AB	AB	AB	AB	AB	MODERATE ASCITIS	4.6	4.3	42	34	2.2	346	4.8	NR	ND
20-617	AB	842163	42/F	AB	AB	AB	AB	AB	AB	AB	AB	ND	5.2	5	256	262	4.2	100	4.2	NR	ANA2+, ASMA 2+, LKM 1+, ANCA 2+
20-1112	BK	886527	37/F	P	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS WITH PORTAL HTN	38.2	26.8	174	116	5.2	65	9	NR	ANA 3+ ASMA3+
20-1199	CL	890059	38/M	AB	AB	AB	AB	AB	AB	AB	AB	COARSE ECHOTEXTURE OF LIVER - CIRRHOSIS	1.42	0.59	110	101	5.2	293	6.5	NR	ANA 3+, LKM 1+, ASMA 2+
20-1205	BK	890705	45/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	1.2	0.4	70	80	4	200	7	NR	ND
20-1617	RL	842163	42/F	AB	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	2.8	0.58	54	49	2.1	77	7	NR	ANA 1+, SMA 3+, LKM 2+
20-1702	P	848034	56/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISAESE	1.3	0.6	93	43	5	246	8	NR	ND
20-2052	JK	901544	20/F	AB	AB	AB	AB	AB	AB	AB	AB	ATROPHY RIGHT LOBE OF LIVER	1.47	0.76	179	71	2.28	160	9.1	NR	ASMA 3+ ANA 2+
20-2547	IK	906031	63/F	AB	P	AB	AB	AB	AB	AB	AB	FATTY LIVER GRADE IV	1.19	0.24	52	53	5	288	5	NR	ANA 2+
20-3017	DP	885952	29/M	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	2	1.3	35	40	2.5	150	5.2	HEP-B POSITIVE	ND
20-3075	BK	910603	49/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISAESE	1.2	0.4	60	72	4	70	8	NR	ASMA 3+, ANA 3+
20-3241	AC	915341	64/F	P	AB	AB	AB	AB	AB	AB	AB	ND	24.56	17	284	252	3.2	162	5.6	HBV-POSITIVE	ANA 1+
20-3451	NK	921262	34/F	AB	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE WITH PORTAL HTN	2.53	1.87	34	21	2.6	250	7.8	NR	AMA 2+, ANA 4+
20-3490	CS	919948	60/M	P	AB	AB	AB	AB	AB	AB	AB	ND	28.7	21.6	50	31	3.2	437	5.3	NR	ND
20-3503	TD	70739	48/F	AB	AB	AB	AB	AB	AB	AB	AB	LIRADS LESION IN RIGHT LOBE OF LINING	10.9	9.7	529	451	2.9	326	5.9	NR	ASMA 2+, ANA 2+
20-3535	NK	922797	36/M	AB	AB	P	AB	AB	AB	AB	AB	HEPATOSPLENOMEGALY WITH GALL BLADDER WALL EDEMA	24.9	20.4	200	210	2.3	127	5.1	NR	ND
20-3693	KS	923705	75/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	1.1	0.56	17	9	2.8	40	5.4	NR	ND
20-3802	SR	925173	40/F	AB	AB	AB	AB	P	AB	AB	AB	LIVER IS DYSMORPHIC- CIRRHOSIS AND PTN	10.5	9.7	617	225	2.13	140	7.6	NR	ASMA 3+, ANA 4+
20-4026	DS	928184	65/M	AB	AB	P	P	AB	P	AB	AB	ND	0.88	0.43	33	25	3.5	115	7.1	NR	ND
20-4039	SK	928661	45/M	AB	AB	P	AB	AB	P	AB	AB	CIRRHOSIS	7.17	5.3	55	53	3.18	122	5.2	NR	NEGATIVE
20-4384	KK	336958	52/F	AB	AB	AB	AB	AB	AB	AB	AB	FATTY LIVER GRADE II	5.55	3.87	96	51	3.7	107	6.9	NR	ANA2+, ASMA 2+, LKM 1+, ANCA 2+
20-4386	DK	914194	55/F	P	AB	AB	AB	AB	AB	AB	AB	CHRONIC LIVER DISEASE	19.5	15.2	90	28	2.24	323	4.2	NR	ANA 2+
20-4580	HS	921743	26/F	P	AB	AB	AB	AB	AB	AB	AB	CIRRHOSIS	17.14	10.72	60	65	4.18	125	7.5	NR	LKM1+
20-4709	TL	934011	65/M	AB	AB	AB	AB	AB	AB	AB	AB	ND	8.5	8	80	82	4.2	250	5.2	NR	ND
20-4736	KM	935300	23/F	P	AB	AB	AB	AB	AB	AB	AB	ND	23	17.26	35	29	3.5	179	6.1	NR	ANA 3+
20-4770	RK	201994	43/M	AB	AB	AB	AB	AB	AB	AB	AB	VENO-OCCLUSIVE DISEASE	3.8	1.31	42	44	3.9	230	8.1	NR	ND

HISTO NO	INITIALS	CERULOPLASMIN	IRON	LIVER BIOPSY GROSS NUMBER	LENGTH	ARCHITECTURE	FATTY CHANGE TYPE	FATTY CHANGE DEGREE
19-11058	NM	ND	ND	3	0.1,0.2 AND 0.4 CM	DISTORTED PORTAL AND LOBULAR ARCHITECURE WITH NODULE FORMATION	AB	AB
19-11278	SK	ND	ND	1	1.5 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION SEPRATED BY THICK SEPTA AND LARGE PORTAL SCAR	AB	AB
19-11395	DD	ND	ND	2	1.4 AND 1.2 CM	DISTORTED LOBULAR ARCHITECTURE WITH NODULE FORMATION	MACROVESICULAR	0.1
19-11647	AKG	ND	ND	3	1,0.5 AND 0.2 CM	DISTORTED PORTAL AND LOBULAR ARCHICTURE WITH NODULE FORMATION	AB	AB
19-12245	SS	ND	ND	2	0.3 AND 0.4 CM	INFILTRATED	AB	AB
19-12257	JR	ND	ND	2	0.5X0.3X0.2 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION THICK AND THIN FIBROSIS SEPTA	MACROVESCULAR STEATOSIS	AB
19-12612	NS	30.5	ND	1	1.2CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION	MACROVESCULAR STEATOSIS	AB
19-12657	AT	24	ND	2	1 AND 1.5 CM	DISTORTED PORTAL AND LOBULAR ARCHICTURE WITH NODULE FORMATION	AB	AB
19-12896	AP	18.3	ND	1	1.2 CM	DISTORTED PORTAL AND LOBULAR ARCHICTURE WITH NODULE FORMATION	AB	AB
19-13088	S	ND	ND	1	1.2 CM	FOCAL LOBULAR DISARRAY	AB	AB
19-13523	A	ND	ND	1	0.6 CM	MAINTAINED PORTAL TO LOBULAR ARCHITECTURE	AB	AB
19-13625	M	ND	ND	1	1.5 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION WITH PRESENCE OF LARGE PORTAL SCAR	AB	AB
19-14106	JS	ND	ND	1	1 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE WITH NODULE FORMATION SEPRATED BY THICK SEPTA	AB	AB
19-14425	MK	ND	ND	2	0.7, 0.8 CM	DISTORTED WITH NODULE FORMATION	AB	AB
'19-14560	BG	17.8	ND	1	1.2 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE WITH NODULE FORMATION SEPRATED BY VARIABLE SIZED SEPTA	AB	AB
19-14837	SM	48.2	ND	1	1 CM	MAINTAINED PORTAL AND LOBULAR ARCHITECTURE	AB	AB
19-15101	AK	ND	ND	1	1 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE WITH NODULE FORMATION	AB	AB
19-15205	BS	ND	ND	1	0.8CM	DILATED PORTAL AND LOBULATED ARCHITECTURE	MACROVESICULAR	0.2
19-15254	CS	ND	ND	1	1.7 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION	AB	AB
19-15470	R	ND	ND	1	1.3 CM	MARKED LOBULAR DISARRAY	AB	AB
19-15479	PS	ND	ND	2	0.8, 0.6 CM	MAINTAINED ARCHITECTURE	AB	AB
19-15531	SS	35.1	ND	2	1.5 AND 1.8 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION SEPERATED BY THICK AND THIN SEPTA	MACROVESCULAR STEATOSIS	0.4
19-15357	VD	ND	ND	1	1.5 CM	MARKED LOBULAR DISARRAY	AB	AB
19-15598	HS	ND	ND	2	0.1, 0.2 CM	DISTORTED ARCHITECTURE WITH NODULE FORMATION	AB	AB
20-67	A	ND	ND	1	1.2 CM	MAINTAINED ARCHITECTURE	AB	AB
20-71	RCS	ND	ND	1	1.2 CM	MARKED LOBULAR DISARRAY	AB	AB
20-353	VS	ND	ND	3	1.5CM	DISTORTED ARCHITECTURE WITH ATTEMPTED NODULE FORMATION	AB	AB
20-617	AB	ND	ND	1	1.2 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.05
20-1112	BK	20	ND	2	0.4 & 1.2 CM	DISTORTED WITH NODULE FORMATIONSEPRATED BY THICK FIBROUS SEPTAL	AB	AB
20-1199	CL	28	ND	2	0.4 & 1 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB
20-1205	BK	ND	ND	1	1.5 CM	DISTORTED WITH PORTO PORTAL SEPTAL AND AN OCCASIONAL SMALL NODULE FORMATION	MACROVESICULAR	0.4
20-1617	RL	NORMAL	ND	1	1.2 CM	DISTORTED WITH NODULE FORMATION	MACROVESICULAR	0.05
20-1702	P	ND	ND	1	1.8 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE WITH NODULE FORMATION SEPRATED BY THICK SEPTAL	MACROVESICULAR	0.2
20-2052	JK	ND	ND	2	1 & 0.4 CM	DISTORTED PORTAL AND LOBULAR ARCHITECTURE WITH NODULE FORMATION	AB	AB
20-2547	IK	26.4	ND	2	1.3 & 1.2	DISTORTED WITH NODULE FORMATION SEPRATED BY THIN FIBROUS SEPTAL	MACROVESICULAR	0.4
20-3017	DP	ND	ND	1	2 CM	DISTORTED WITH OCCASIONAL NODULE FORMATION	AB	AB
20-3075	BK	14	ND	1	2 CM	DISTORTED WITH THIN PORTAO PORTAL SEPTAL	AB	AB
20-3241	AC	20	ND	2	0.8 & 1.2 CM	MARKED LOBULAR DISARRAY	AB	AB
20-3451	NK	ND	ND	1	1.5 CM	DISTORTED WIT NODULE FORMATION AND PORTAL SCAR	AB	AB
20-3490	CS	ND	ND	1	1.8 CM	MODERATE LOBULAR DISARRAY	AB	AB
20-3503	TD	10	ND	1	1 CM	MARKED LOBULAR DISARRAY	AB	AB
20-3535	NK	36	ND	1	1.6 CM	MARKED LOBULAR DISARRAY	MACROVESICULAR	0.4
20-3693	KS	ND	52	MULTIPLE	2X1.5X1 CM	NORMAL	AB	AB
20-3802	SR	15.6	ND	1	1.5 CM	MARKED LOBULAR DISARRAY	AB	AB
20-4026	DS	ND	ND	1	0.4 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	MACROVESICULAR	0.4
20-4039	SK	ND	ND	1	1.8 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION SEPRATED WITH VARIABLY SIZED FIBROUS SEPTAL	AB	AB
20-4384	KK	11.9	ND	1	1.4 CM	DISTORTED WITH NODULE FORMATION BY VARIABLY SIZED SEPTAL	AB	AB
20-4386	DK	24.3	ND	1	1.8 CM	MAINTAINED	AB	AB
20-4580	HS	22	ND	1	1.5 CM	DISTORTED WITH NODULE FORMATION SEPRATED BY THICK FIBROUS SEPTAL AND PORTAL SCAR	MACROVESICULAR	0.2
20-4709	TL	ND	ND	MULTIPLE	0.2 TO 0.8 CM	AB	AB	AB
20-4736	KM	22	ND	12	0.5 TO 1.2 CM	MAINTAINED	AB	AB
20-4770	RK	ND	ND	1	1 CM	DISTORTED WITH ATTEMPTED NODULE FORMATION	AB	AB

HISTO NO	INITIALS	CHOLESTASIS	BILE	BILE DUCT	MALLORY DENK BODIES
19-11058	NM	INTRACELLULAR	BILE PLUGS	ABSENT	P
19-11278	SK	AB	AB	ABSENT	AB
19-11395	DD	AB	AB	ABSENT	AB
19-11647	AKG	AB	AB	ABSENT	AB
19-12245	SS	AB	AB	ABSENT	AB
19-12257	JR	AB	AB	ABSENT	P
19-12612	NS	P	AB	ABSENT	AB
19-12657	AT	BILE PLUG	AB	ABSENT	AB
19-12896	AP	AB	AB	PROLIFERATION	AB
19-13088	S	FOCAL	BILE PLUGS	A	AB
19-13523	A	AB	AB	ABSENT	AB
19-13625	M	AB	AB	PROLIFERATION	AB
19-14106	JS	INTRAHEPATIC	AB	ABSENT	AB
19-14425	MK	AB	AB	REACTION	AB
'19-14560	BG	AB	AB	ABSENT	AB
19-14837	SM	CHOLESTASIS CANNALICULAR TYPE WITH BILE PLUGS AT PERIVENULAR AREA	AB	ABSENT	AB
19-15101	AK	INTRACANALICULAR	AB	ABSENT	AB
19-15205	BS	AB	AB	BSENT	AB
19-15254	CS	INTRAHEPATIC CHOLESTASIS	AB	PROLIFERATION	AB
19-15470	R	P	BILE PLUGS	ABSENT	AB
19-15479	PS	AB	AB	ABSENT	AB
19-15531	SS	AB	AB	ABSENT	AB
19-15357	VD	INTRAHEPATIC CHOLESTASIS	BILE PLUGS	ABSENT	AB
19-15598	HS	AB	AB	ABSENT	P
20-67	A	AB	AB	ABSENT	AB
20-71	RCS	P	AB	ABSENT	AB
20-353	VS	AB	AB	ABSENT	AB
20-617	AB	AB	AB	PRESENT	AB
20-1112	BK	P	AB	P	AB
20-1199	CL	AB	AB	AB	AB
20-1205	BK	AB	AB	AB	AB
20-1617	RL	P	AB	AB	AB
20-1702	P	AB	AB	AB	AB
20-2052	JK	AB	AB	PRESENT	AB
20-2547	IK	AB	AB	AB	AB
20-3017	DP	AB	AB	AB	AB
20-3075	BK	AB	AB	AB	AB
20-3241	AC	INTRACELLULAR AND PERIVENULAR	AB	AB	AB
20-3451	NK	AB	AB	AB	AB
20-3490	CS	CANALICULAR CHOLESTASIS IN CENTRI LOBULAR AREAS	BILE PLUGS	AB	AB
20-3503	TD	P	AB	AB	AB
20-3535	NK	PERICANALICULAR TYPE	BILE PLUGS	AB	AB
20-3693	KS	AB	AB	AB	AB
20-3802	SR	INTRACELLULAR AND CANALICULAR TYPE	BILE PLUGS	AB	AB
20-4026	DS	AB	AB	AB	AB
20-4039	SK	PROMINENT	BILE PLUGS	AB	P
20-4384	KK	AB	AB	AB	AB
20-4386	DK	P	AB	AB	AB
20-4580	HS	CANALICULAR AND INTRA CELLULAR TYPE	BILE PLUGS	AB	AB
20-4709	TL	AB	AB	AB	AB
20-4736	KM	CANALICULAR AND CENTRI LOBULAR AREA	AB	PRESENT	AB
20-4770	RK	AB	AB	AB	AB

HISTO NO	INITIALS	HEPATOCYTES
19-11058	NM	MILD BALLOONING DEGENERATION
19-11278	SK	MILD BALLOONING DEGENERATION
19-11395	DD	MILD BALLOONING DEGENERATION
19-11647	AKG	MODERATE BALLOONING DEGENRATION
19-12245	SS	LIVER BIOPSY SHOWS PRESENCE OF MANY HEPATOCYTES WHICH ARE COMPRESSED BY LARGE AMOUNT OF OF EXTRACELLULAR , A CELLULAR MATERIAL WHICH IS HIGHLIGHTED O CONGO-RED – AMYLOID
19-12257	JR	MILD BALLOONING DEGENERATION
19-12612	NS	MILD BALLOONING DEGENERATION
19-12657	AT	MILD BALLOONING DEGENERATION
19-12896	AP	MILD BALLOONING DEGENERATION
19-13088	S	ACIDOPHILIC BODIES SEEN
19-13523	A	PRESENCE OF BROWN PIGMENT IN PERIVENULAR AREA
19-13625	M	MILD BALLOONING DEGENERATION
19-14106	JS	MILD BALLOONING DEGENERATION
19-14425	MK	MILD BALLOONING DEGENERATION
'19-14560	BG	MILD BALLOONING DEGENERATION
19-14837	SM	MILD BALLOONING DEGENERATION
19-15101	AK	EMPERPOLESIS
19-15205	BS	MILD BALLOONING DEGENRATION
19-15254	CS	MILD BALLOONING DEGENERATION
19-15470	R	ROSETTE FORMATION AND GIANT CELL TRANSFORMATION AND MARKED FEATHRY DEGENERATION
19-15479	PS	UR
19-15531	SS	MILD BALLOONING DEGENERATION
19-15357	VD	CLD
19-15598	HS	MODERATE BALLOONING DEGENRATION
20-67	A	UR
20-71	RCS	MILD BALLOONING DEGENERATION
20-353	VS	MILD BALLOONING DEGENERATION
20-617	AB	MILD BALLOONING DEGENERATION
20-1112	BK	MILD BALLOONING DEGENERATION
20-1199	CL	MILD BALLOONING DEGENERATION
20-1205	BK	MILD BALLOONING DEGENERATION
20-1617	RL	MILD BALLOONING DEGENERATION
20-1702	P	MILD BALLONING DEGENERATION OF HEPATOCYTES
20-2052	JK	BALLONING DEGENERATION AND ENPERIPOLESIS OF HEPATOCYTES
20-2547	IK	MILD BALLOONING DEGENERATION
20-3017	DP	MILD BALLONING DEGENERATION
20-3075	BK	MILD BALLONING DEGENERATION
20-3241	AC	MILD BALLOONING DEGENERATION
20-3451	NK	MILD BALLONING DEGENERATION
20-3490	CS	MILD BALLOONING DEGENERATION
20-3503	TD	MANY ACIDOPHIIC BODIES WITH HEPATIC ROSETTING
20-3535	NK	MILD BALLOONING DEGENRATION
20-3693	KS	WELL FORMED EPITHELIOD GRANULOMAS
20-3802	SR	ROSETTING NOTED
20-4026	DS	MILD BALLOONING DEGENRATION CHRONIC INFLAMMATORY INFILTRATE
20-4039	SK	MILD BALLOONING DEGENERATION
20-4384	KK	BALLONING DEGENERATION
20-4386	DK	BALLONING DEGENERATION
20-4580	HS	MILD BALLOONING AND ROSSETING
20-4709	TL	PRESENCE OF PINK ACELLULAR EOSINOPHILIC MATERIAL AROUND THE SINUSOID COMPRESSING THE ADJACENT HEPATOCYTES CAUSING ATROPHY
20-4736	KM	BALLONING DEGENERATION
20-4770	RK	CENTRI LOBULAR HEPATOCYTES SHOW ATROPHIC CHANGES WITH FOCAL DROP OUT. ZONE 2 AND ZONE 3 AREAS SHOW PERISINUSOIDAL FIBROSIS SURROUNDING THE DILATED SINUSOIDS. HEPATOCYTES REGENERATIVE CHANGES

HISTO NO	INITIALS	INFLAMMATORY INFILTRATE PORTAL TRACTS/SEPTA/SINUSOIDS	PLASMA CELL RICH	GRANULOMA	INTERFACE HEPATITIS	SPOTTY HEPATIC NECROSIS
19-11058	NM	MODERATE INFILTRATION BY NEUTROPHILS, LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
19-11278	SK	DENSE INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, EOSINOPHILS, NEUTROPHILS, PLASMA CELLS AND HISTIOCYTES	AB	AB	P	AB
19-11395	DD	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES.	AB	AB	AB	AB
19-11647	AKG	ABSENT	AB	AB	AB	AB
19-12245	SS	MILD CHRONIC INFLAMMATORY INFILTRATES	AB	AB	AB	AB
19-12257	JR	MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE AS LYMPHOCYTES, HISTIOCYTES, FEW NEUTROPHILS	AB	AB	AB	AB
19-12612	NS	MIXED INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES , NEUTROPHILS AND EOSINOPHILS	AB	AB	P	AB
19-12657	AT	MODERATE INFILTRATION BY MIXED INFILTRATE COMPRISING OF LYMPHOCYTES, HISTIOCYTES, NEUTROPHILS AND FEW EOSINOPHILS	AB	AB	FOCAL	AB
19-12896	AP	DENSE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
19-13088	S	MODERATDE INFILTRATION BY LYMPHOCYTES, HISTIOCYTES AND EOSINOPHILS	AB	AB	HEPATITIS NOTED	AB
19-13523	A	UNREMARKABLE	AB	AB	AB	AB
19-13625	M	INFILTRATED BY DENSE INFLAMMATORY INFILTRATE COMPOSED OF LYMPHOCYTES, HISTIOCYTES, FEW NEUTROPHILS AND EOSINOPHILS	AB	AB	P	AB
19-14106	JS	MODERATE INFILTRATE BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND FEW PLASMA CELLS	AB	AB	AB	AB
19-14425	MK	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELL SEEN AS LYMPHOCYTES, FEW PLASMA CELLS AND EOSINOPHILS	AB	AB	AB	AB
'19-14560	BG	MODERATE INFILTRATION BY MIXED INFLAMMATORY INFILTRATION SEEN AS LYMPHOCYTES, HISTIOCYTES AND FEW PLASMA CELLS	AB	AB	P	P
19-14837	SM	MILD INFILTRATION BY MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND FEW NEUTROPHILS	AB	AB	AB	AB
19-15101	AK	DENSE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND FEW PLASMA CELLS	AB	AB	PRESENT	AB
19-15205	BS	MODERAQTE INFILTRATION BY LYMPHOCYTES, PLASMA CELLS AND FEW HISTIOCYTES	AB	AB	PRESENT	AB
19-15254	CS	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELLS	AB	AB	P	AB
19-15470	R	MILD CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES	AB	AB	AB	AB
19-15479	PS	MILD INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND FEW EOSINOPHILS WITH FOCAL EDEMA	AB	AB	AB	AB
19-15531	SS	MILD INFILTRATION BY LYMPHOCYTES, HISTIOCYTES	AB	AB	AB	AB
19-15357	VD	DENSE INFILTRATION BY LYMPHOCYTES, HISTIOCYTES, NEUTROPHILS AND MANY EOSINOPHILS	AB	AB	AB	P
19-15598	HS	MILD TO MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES.	AB	AB	AB	AB
20-67	A	ABSENT	AB	AB	AB	AB
20-71	RCS	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND EOSINOPHILS	AB	AB	AB	AB
20-353	VS	MILD INFILTRATION BY MONONUCLEAR CELLS COMPOSED OF LYMPHOCYTES, OCCASIONAL PLASMA CELLS AND FEW EOSINOPHILS	AB	AB	AB	AB
20-617	AB	MODERATE DENSE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES, PLASMA CELLS AND EOSINOPHILS	AB	AB	P	AB
20-1112	BK	MODERATE TO DENSE INFILTRATION BY ACUTE AND CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, PLASMA CELLS, HISTIOCYTES AND NEUTROPHILS	AB	AB	P	P
20-1199	CL	INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	AB
20-1205	BK	MILD CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND HISTIOCYTES	AB	AB	AB	AB
20-1617	RL	DENSE MIXED MILD INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES, PLASMA CELLS, NEUTROPHILS AND FEW EOSINOPHILS	AB	AB	P	AB
20-1702	P	DENSE ACUTE AND CHRONIC INFLAMMATORY CELLS SEEN AS NEUTROPHILS, LYMPHOCYTES AND HISTIOCYTES.	AB	AB	P	P
20-2052	JK	DENSE INFILTRATION BY CHRONIC INFLAMMATORY CELLS SEEN AS PLASMA CELLS, LYMPHOCYTES, HISTIOCYTES AND FEW NEUTROPHILS	AB	AB	P	P
20-2547	IK	MODERATE INFILTRATION BY CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES AND FEW EOSINOPHILS	AB	AB	P	P
20-3017	DP	MILD CHRONIC INFILTRATE	AB	AB	AB	P
20-3075	BK	CHRONIC INFLAMMATORY CELLS SEEN AS PLASMA CELLS, EOSINOPHILS, LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	P
20-3241	AC	MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES, EOSINOPHILS AND HISTIOCYTES	AB	AB	P	P
20-3451	NK	MILD CHRONIC INFLAMMATORY INFILTRATES	AB	AB	P	P
20-3490	CS	EXPANDED AND INFILTRATED BY MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, HISTIOCYTES AND FEW EOSINOPHILS	AB	AB	P	P
20-3503	TD	MODERATE TO DENSE MIXED LOBULAR INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES, HISTIOCYTES, NEUTROPHILS AND FEW EOSINOPHILS	AB	AB	P	P
20-3535	NK	MILD INFILTRATION BY MIXED INFLAMMATORY INFILTRATE	AB	AB	AB	AB
20-3693	KS	MILD ACUTE AND CHRONIC INFLAMMATORY INFILTRATE	AB	P	AB	P
20-3802	SR	DENSE INFILTRATION BY MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES, EOSINOPHILS, HISTIOCYTES AND FEW PLASMA CELLS	AB	AB	P	AB
20-4026	DS	MILD CHRONIC INFLAMMATORY INFILTRATES	AB	AB	AB	AB
20-4039	SK	FOCI OF MIXED INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, HISTIOCYTES AND PLASMA CELLS	AB	AB	P	P
20-4384	KK	DENSE CHRONIC INFLAMMATORY INFILTRATE SEEN AS PLASMA CELLS, LYMPHOCYTES AND HISTIOCYTES	AB	AB	P	P
20-4386	DK	DUCTOPENIA WITH PERIDUCTAL AND PORTAL FIBROSIS	AB	AB	AB	AB
20-4580	HS	DENSE INFILTRATION BY ACUTE AND CHRONIC INFLAMMATORY INFILTRATE SEEN AS NEUTROPHILS, LYMPHOCYTES, FEW PLASAM CELLS AND OCCASIONAL EOSINOPHILS	AB	AB	P	P
20-4709	TL	MILD CHRONIC INFLAMMATORY INFILTRATES	AB	AB	AB	AB
20-4736	KM	MILD CHRONIC INFLAMMATORY INFILTRATE SEEN AS LYMPHOCYTES AND EOSINOPHILS	AB	AB	AB	P
20-4770	RK	SHOW INCREASED FIBROSIS WITH PROTAL VIEW CONTRI LOBULAR SHOW PERIVENULAR SINUCOIDAL SILATION AROUND IT. CONGESTION OF SINUSOID ALSO SEN WITH SPILL ONCE OF RED BLOOD CELLS INTO HEPATOCYTIC PLATES	ILATION AROUND IT. CO	AB	AB	AB

HISTO NO	INITIALS	CONFLUENT NECROSIS	FIBROSIS	STAINS		
				IRON	MASSON TRICHOME	RETICULIN
19-11058	NM	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-11278	SK	AB	GRADE IV	FOCALLY POSITIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-11395	DD	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-11647	AKG	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION SEPRATED WITH THICK AND THIN	MAINTAINED
19-12245	SS	AB	AB	ND	ND	ND
19-12257	JR	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION SEPTARED WITH THICK AND THIN	MAINTAINED
19-12612	NS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-12657	AT	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-12896	AP	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-13088	S	AB	GRADE II	NEGATIVE	DO NOT HIGHLIGHTS INCREASED FIBROSIS	FOCAL COLLAPSE
19-13523	A	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS FIBROSIS	MAINTAINED
19-13625	M	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-14106	JS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-14425	MK	AB	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
'19-14560	BG	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-14837	SM	AB	AB	NEGATIVE	HIGHLIGHTS MINIMAL TO FOCAL PERICELLULAR	MAINTAINED
19-15101	AK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION SEPRATED BY SEPTAL	MAINTAINED
19-15205	BS	P	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION	MAINTAINED
19-15254	CS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
19-15470	R	AB	AB	NEGATIVE	HIGHLIGHTS SIGNIFICANT FIBROSIS	MAINTAINED
19-15479	PS	AB	AB	NEGATIVE	DO NOT HIGHLIGHTS FIBROSIS	MAINTAINED
19-15531	SS	AB	GRADE II	NEGATIVE	HIGHLIGHTS PERICELLULAR FIBROSIS AND NODULE FORMATION	MAINTAINED
19-15357	VD	AB	AB	NEGATIVE	HIGHLIGHT FOCAL INCREASE IN PORTAL FIBROSIS	MAINTAINED
19-15598	HS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-67	A	AB	AB	NEGATIVE	DO NOT HIGHLIGHTSFIBROSIS	MAINTAINED
20-71	RCS	AB	GRADE I	NEGATIVE	HIGHLIGHTS FOCAL PERICELLULAR FIBROSIS	MAINTAINED
20-353	VS	AB	GRADE III	FOCAL POSITIVITY IN KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION AND PERICELLULAR	MAINTAINED
20-617	AB	AB	GRADE IV	HIGHLIGHTS HEMOSIDERIN IS KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION SEPRATED BY SEPTAL	MAINTAINED
20-1112	BK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION WITH FIBROSIS	MAINTAINED
20-1199	CL	AB	GRADE III	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-1205	BK	AB	GRADE III	NEGATIVE	HIGHLIGHTS ARE ATTEMPTED NODULE WITH PORTO PORTAL SEPTAL AND FOCAL PERICELLULAR FIBROSIS	MAINTAINED
20-1617	RL	AB	GRADE IV	HIGHLIGHTS HEMOSIDERIN IS KUPFFER CELLS	HIGHLIGHTS NODULE FORMATION SEPRATED BY SEPTAL	MAINTAINED
20-1702	P	AB	GRADE II	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND PERICELLULAR FIBROSIS	MAINTAINED
20-2052	JK	AB	GRADE IV	POSITIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-2547	IK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION SEPRATED BY FIBROUS SEPTAL AND PERICELLULAR AND PERIVENULAR FIBROSIS	MAINTAINED
20-3017	DP	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-3075	BK	ESTION WITH H	GRADE III	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS WITH THIN PERIPORTAL SEPTAL	MAINTAINED
20-3241	AC	AB	GRADE II	NEGATIVE	FOCAL PORTAL FIBROSIS	COLLAPSE
20-3451	NK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-3490	CS	AB	ND	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	MAINTAINED
20-3503	TD	AB	GRADE II	NEGATIVE	HIGHLIGHTS FOCAL INCREASE IN PORTAL FIBROSIS	COLLAPSE
20-3535	NK	AB	GRADE II	NEGATIVE	HIGHLIGHTS PROMINENT PERICELLULAR FIBROSIS	COLLAPSE
20-3693	KS	AB	NA	NEGATIVE	ND	MAINTAINED
20-3802	SR	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-4026	DS	AB	GRADE III	NEGATIVE	HIGHLIGHTS ATTEMPTED NODULE FORMATION AND PERICELLULAR FIBROSIS	MAINTAINED
20-4039	SK	P	GRADE III	NEGATIVE	ATTEMPTED NODULE AND PERICELLULAR AND PERIVENULAR FIBROSIS	HIGHLIGHTS RETICULIN RICH AREA IN THE NODULE
20-4384	KK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION	MAINTAINED
20-4386	DK	AB	GRADEII	NEGATIVE	HIGHLIGHTS PORTAL FIBROSIS WITH FOCAL PERIPORTAL FORMATION	MAINTAINED
20-4580	HS	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION SEPRATED BY FIBROUS SEPTAL AND PERICELLULAR FIBROSIS	HIGHLIGHTS RETICULIN RICH AREA IN THE NODULE
20-4709	TL	AB	AB	NEGATIVE	AB	MAINTAINED
20-4736	KM	AB	AB	NEGATIVE	DO NOT HIGHLIGHT FIBROSIS	HIGHLIGHTS RETICULIN RICH AREA
20-4770	RK	AB	GRADE IV	NEGATIVE	HIGHLIGHTS NODULE FORMATION AND PERIVENULAR AND PERI SINUSOIDAL FIBROSIS	HIGHLIGHTS RETICULIN RICH AREA

HISTO NO	INITIALS	OTHERS
19-11058	NM	ND
19-11278	SK	ND
19-11395	DD	ND
19-11647	AKG	ND
19-12245	SS	ND
19-12257	JR	ND
19-12612	NS	COPPER PIGMENT IN PERSEPTAL AREA
19-12657	AT	RHODAMINE STAIN HIGHLIGHTS ACCUMULATION OF COPPER PIGMENT BOTH IN PERISEPTAL AREA AND IN THE CENTRILOBULAR HEPATOCYTES
19-12896	AP	ND
19-13088	S	ND
19-13523	A	ND
19-13625	M	ND
19-14106	JS	ND
19-14425	MK	ND
'19-14560	BG	ND
19-14837	SM	ND
19-15101	AK	ND
19-15205	BS	ND
19-15254	CS	ND
19-15470	R	ND
19-15479	PS	PAS GMS FOR FUNGUS ARE NEGATIVE
19-15531	SS	ND
19-15357	VD	ND
19-15598	HS	ND
20-67	A	ND
20-71	RCS	ND
20-353	VS	ND
20-617	AB	ND
20-1112	BK	ND
20-1199	CL	ND
20-1205	BK	ND
20-1617	RL	ND
20-1702	P	ORCEIN AND RHODENINE FOR COPPER NEGATIVE
20-2052	JK	ND
20-2547	IK	ND
20-3017	DP	ND
20-3075	BK	ND
20-3241	AC	ND
20-3451	NK	ND
20-3490	CS	ND
20-3503	TD	ND
20-3535	NK	ND
20-3693	KS	20% ZN STAIN FOR AFB, PAS AND GMS STAIN FOR FUNGUS ARE NEGATIVE
20-3802	SR	ND
20-4026	DS	ND
20-4039	SK	ND
20-4384	KK	ORCEIN AND RHODENINE FOR COPPER IS NEGATIVE
20-4386	DK	RHODANINE STAIN FOR COPPER IS NEGATIVE
20-4580	HS	ND
20-4709	TL	METHYLE VIOLET STAIN AND CONGO RED STAIN FOR AMYLOID IS POSITIVE
20-4736	KM	ND
20-4770	RK	ND

HISTO NO	INITIALS	IMPRESSION
19-11058	NM	NASH GRADE 4 STAGE 4
19-11278	SK	AUTOIMMUNE HEPATITIS
19-11395	DD	NASH GRADE 4 SATGE 4
19-11647	AKG	NASH GRADE 2 STAGE 4
19-12245	SS	AMYLOIDOSIS
19-12257	JR	CIRRHOSIS- IDIOPATHIC
19-12612	NS	CIRRHOSIS- IDIOPATHIC
19-12657	AT	CIRRHOSIS- ? WILSON'S DISEASE
19-12896	AP	CIRRHOSIS- IDIOPATHIC
19-13088	S	ACUTE HEPATITIS(HEV POSITIVE)
19-13523	A	NO SPECIFIC PATHOLOGY- R/O GILBERT SYNDROME
19-13625	M	PRIMARY BILIARY CIRRHOSIS
19-14106	JS	NASH GRADE 2 STAGE 4
19-14425	MK	NASH GRADE 3 STAGE 4
'19-14560	BG	PRIMARY BILIARY CIRRHOSIS
19-14837	SM	DILI
19-15101	AK	AUTOIMMUNE HEPATITIS
19-15205	BS	ASH GRADE 3 STAGE 4
19-15254	CS	LARGE DUCT OBSTRUCTION
19-15470	R	NEONATEL GIANT CELL HEPATITIS
19-15479	PS	NO SPECIFIC PATHOLOGY
19-15531	SS	NASH GRADE 3 STAGE 4
19-15357	VD	DILI (LINEZOLID AND IMIPENUM)
19-15598	HS	NASH GRADE 4 STAGE 4
20-67	A	NO SPECIFIC PATHOLOGY
20-71	RCS	DILI (HERBAL MEDICINES)
20-353	VS	DILI (LEVOFLOXACIN, PR TRANSPLANT)
20-617	AB	AUTOIMMUNE HEPATITIS
20-1112	BK	CIRRHOSIS (PRIMARY BILIARY CIRRHOSIS AND AUTOIMMUNE HEPATITIS)- OVERLAP SYNDROME
20-1199	CL	AUTOIMMUNE HEPATITIS
20-1205	BK	NASH GRADE 4 STAGE 4
20-1617	RL	AUTOIMMUNE HEPATITIS
20-1702	P	NASH GRADE 4 STAGE 4
20-2052	JK	AUTOIMMUNE HEPATITIS
20-2547	IK	NASH GRADE 5 STAGE 4
20-3017	DP	CHRONIC VIRAL HEPATITIS (HBV POSITIVE) WITH MILD ACTIVITY HAI SCORE 3, GRADE I, STAGE 3 .
20-3075	BK	AUTOIMMUNE HEPATITIS
20-3241	AC	DILI (HERBAL MEDICINES)
20-3451	NK	PRIMARY BILIARY CIRRHOSIS
20-3490	CS	DILI (ATT)
20-3503	TD	DILI (HERBAL MEDICINES)
20-3535	NK	ASH GRADE 3 STAGE 1
20-3693	KS	GRANULOMATOUS HEPATITIS- TUBERCULOSIS
20-3802	SR	AUTOIMMUNE HEPATITIS
20-4026	DS	ASH GRADE 4 STAGE 4
20-4039	SK	ASH GRADE 6 STAGE 4
20-4384	KK	AUTOIMMUNE HEPATITIS
20-4386	DK	AUTOIMMUNE HEPATITIS
20-4580	HS	NASH GRADE 5 STAGE 4
20-4709	TL	AMYLOIDOSIS
20-4736	KM	AUTOIMMUNE HEPATITIS
20-4770	RK	CHRONIC LIVER DISEASE GOING ON THE CIRRHOSIS WITH UNDERLYING VENO OCCLUSIVE DISEASE- FEATURES OF CHRONIC BUDD-CHIARI SYNDROME

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The Principal,
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Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.05.2019 has approved the plan of thesis of **Navita Gupta**, a student of your college for **MD Pathology** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis

Histopathological spectrum of non neoplastic liver diseases

Supervisor
Dr. Neena Sood
Prof. & Head,
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DMCH, Ludhiana.

Co-Supervisor
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The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

69. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
70. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

HISTOPATHOLOGICAL SPECTRUM OF NON NEOPLASTIC LIVER DISEASES

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

Doctor of Medicine

in

PATHOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

August, 2018

Dr. NAVITA GUPTA

DEPARTMENT OF PATHOLOGY
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA
LUDHIANA, PUNJAB-141001

ABSTRACT OF PLAN OF THESIS

Title	HISTOPATHOLOGICAL SPECTRUM OF NON NEOPLASTIC LIVER DISEASES
For the degree of	M.D. Pathology
Name of the candidate	Dr. Navita Gupta
Supervisor	Dr. Neena Sood, Professor & Head ,Department of Pathology, DMCH, Ludhiana
Co-supervisor	Dr. Aminder Singh, Assistant Professor , Department of Pathology, DMCH, Ludhiana Dr. Ajit Sood, Professor & Head, Department of Gastroenterology, DMCH, Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: This will be retrospective and prospective study conducted in the department of pathology, Dayanand Medical College & Hospital, Ludhiana. All the cases suspected of non neoplastic liver diseases will be analysed with respect to microscopy and correlated clinically.

Aim of study: Histopathological spectrum of non neoplastic liver diseases.

Materials and methods: All the liver biopsies will be received in container containing 10% formalin. Tissue will be processed and stained with haematoxylin and eosin stain and special stains like masson trichome, reticulin, Periodic Acid Schiff (PAS), prussian blue, rodanine, orcein stains will be performed. The slides will be subjected to histopathological examination under both low power (10X) and high power (40X).

Clinical significance: To obtain of reasonable good database for a more accurate assessment and understanding of etiopathogenesis and diagnosis of non neoplastic liver diseases.

Keywords: Liver biopsy, hepatitis, metabolic liver diseases, cirrhosis.

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"HISTOPATHOLOGICAL SPECTRUM OF NON NEOPLASTIC LIVER DISEASES"** exist in the Department of Pathology at Dayanand Medical College and Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate herself under our supervision and guidance.

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Place : _____

Date : _____

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

Name of candidate	Dr. Navita Gupta
Department	Pathology
Topic of Thesis	HISTOPATHOLOGICAL SPECTRUM OF NON NEOPLASTIC LIVER DISEASES
Likely date of appearing in PG examination	MAY-JUNE 2019
Date of enrollment	MAY 10, 2018
Name of Head of Department	DR.Neena Sood
1) Supervisor **2) Co-Supervisor**	DR. Neena Sood DR. Aminder Singh Dr. Ajit Sood
Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4. HOD Medicine	4. HOD SPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HOD Obst. & Gynae
	8. Legal Expert
	9. Secretary, IEC
	10. Dean Academics
	11. Vice Principal
	12. Principal

Approved: Yes / No

Chairperson
Research Committee

Chairperson
Research Committee

Approved: Yes / ~~No~~

Chairperson
Ethics Committee

Chairperson
Ethics Committee

BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

1	NAME OF THE CANDIDATE AND ADDRESS	Dr. Navita Gupta H/NO. 50, Saraswati Vihar, Jodhamal Road, Hoshiarpur-146001
2	NAME OF THE INSTITUTE	Dayanand Medical College & Hospital, Ludhiana
3	COURSE OF STUDY AND SUBJECT	MD PATHOLOGY
4	DATE OF ADMISSION OF COURSE	10th MAY,2018
5	TITLE OF THE TOPIC	HISTOPATHOLOGICAL SPECTRUM OF NON NEOPLASTIC LIVER DISEASES

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Percutaneous, ultrasound-guided liver biopsy remains the most reliable way to diagnose liver diseases because the method is simple, rapid, inexpensive, and quite safe.[1] Sometimes wedge liver biopsies are taken for unexpected lesions encountered during surgery.

Liver biopsies are done to establish the diagnosis, for staging and/or prognostication, and for treatment planning. The various diseases like viral hepatitis and non viral liver diseases like autoimmune hepatitis, autoimmune cholestasis, drug induced, fatty liver diseases related to alcoholic and non alcoholic causes, metabolic diseases like haemchromatosis, wilson's disease, alpha 1 antitrypsin deficiency require histopathological correlation. Biopsies are also sometimes performed for acute liver failure, liver transplantation rejection and reinfection, solitary and multiple non neoplastic focal hepatic lesions.

For patients with viral hepatitis, liver biopsy is used for grading, staging and for treatment monitoring over the course of disease. Hepatitis A and hepatitis E are the causes of acute hepatitis and hepatitis B, hepatitis C, hepatitis D have the potential to cause chronic disease. The inflammatory cells in both acute and chronic are mainly T lymphocytes. Hence it is the pattern of injury that is different between the two time courses, not the nature of infilterate. In patients with cholestatic liver diseases, liver biopsy is a valuable means of detecting or ruling out the autoimmune disorders when the laboratory findings are not conclusive. Histological examination helps to differentiate alcoholic from non-alcoholic fatty-liver disease and in assessment of inflammation and

degree of fibrosis. There are three forms of alcoholic liver injury namely hepatocellular steatosis, alcoholic steatohepatitis and steatofibrosis. When the laboratory findings are inconclusive liver biopsy can provide the initial diagnosis of storage diseases and metabolic diseases of liver. Among all diagnostic methods for hepatic nodules, liver biopsy has the greatest sensitivity and specificity with respect to the determination of malignancy. Liver-cell adenoma is sometimes difficult to distinguish from well-differentiated hepatocellular carcinoma. In such cases, a biopsy is advisable if the imaging studies are inconclusive. In case of acute and chronic liver allograft rejection, histology remains the gold standard investigation. Acute rejection occurs in 10 to 50 days after the haematopoietic stem cell transplantation. Chronic rejection occurs usually more than 100 days after the transplantation.

Hepatic morphology may be strikingly affected by circulatory disorders. It can be due to impaired blood inflow due to portal vein obstruction and intra or extrahepatic thrombosis. There can be impaired intrahepatic blood flow due to cirrhosis and sinusoidal occlusion. Hepatic vein outflow obstruction can occur due to hepatic vein obstruction (Budd-Chiari syndrome), sinusoidal obstructive syndrome leading to elevated aminotransferases and jaundice.

Prior to performance of liver biopsy, patients should be educated about their liver disease and about investigations other than liver biopsy (if any) that may also provide diagnostic and prognostic information.

6.2 REVIEW OF LITERATURE

Histological assessment of the liver, and thus, liver biopsy, is a cornerstone in the evaluation and management of patients with liver pathology particularly chronic liver disease and is considered to be an integral component of the clinician's diagnostic armamentarium.

Liver biopsies can be divided into two categories based on diameter of biopsy needle: (a) large needle biopsies with a minimum core diameter of 1.0 mm (e.g. Menghini), which are used for the investigation of diffuse diseases (b) thin needle biopsies with an internal diameter of less than 1.0 mm, which are more often employed for the investigation of focal lesions.[2-6]

Liver biopsy currently has three major roles: (a) for diagnosis of multiple parenchymal liver diseases, abnormal liver tests of unknown etiology, fever of unknown origin, focal or diffuse abnormalities on imaging studies (b) for assessment of prognosis (disease staging), and/or (c) to assist in making therapeutic management decisions. Liver histology and clinical and laboratory data are used in conjugation to make the diagnosis.

Based on the lobular model normal liver is divided into lobules that are oriented around hepatic veins with portal tract at lobule's periphery. In the lobule hepatocytes are arranged in sheets or plates in between which lie the sinusoids. These sinusoids are lined by endothelial cells to which are attached the kupffer cells. Stellate cells are present in space of Disease. Liver is vulnerable primarily to viral hepatitis, nonalcoholic and alcoholic liver disease and hepatocellular carcinoma. Hepatic damage can also occur secondary to heart failure, disseminated cancer and extrahepatic infections. Usually hepatic decompensation occurs after weeks, months or many years after onset of

injury. Hepatocytes undergo reversible changes like steatosis and cholestasis. When injury is not reversible hepatocytes die by necrosis or apoptosis. Hepatocytes have stem cell like ability due to which hepatocyte replenishment occurs even when there is significant confluent necrosis. Hence 80-90% of hepatic functional capacity should be lost before hepatic failure ensues. This is the reason that hepatic injury may be imperceptible to the patient and detectable only by abnormal laboratory tests. Hence need for liver biopsy occur mainly in chronic liver diseases.

Liver biopsy has main role in diseases like chronic viral hepatitis B and viral hepatitis C, autoimmune hepatitis, primary biliary cirrhosis, primary sclerosing cholangitis, alcoholic steatohepatitis, metabolic diseases like non alcoholic fatty liver disease, haemchromatosis, wilson's disease, alfa 1 antitrypsin deficiency, liver transplantation rejection/reinfection, solitary and multiple focal hepatic lesions, structural abnormalities of biliary tree like choledochal cyst and fibropolycystic disease, circulatory disorders like hepatic artery compromise, portal vein obstruction, hepatic vein obstruction called as budd chiari syndrome.

According to WHO, 2 billion population of the entire world is infected with hepatitis B out of which India harbors 10–15% of the entire pool.[7] In most studies the overall rate of HBsAg positivity has been reported to range between 2% and 8%.[8,9] In viral hepatitis liver biopsy is used for confirmation of clinical diagnosis, grading the necroinflammatory activity, and staging the extent of fibrosis. Viral hepatitis is caused by hepatotropic virus A, B, C, D, E. Both acute and chronic hepatitis evoke a lymphoplasmacytic infiltrate. Acute viral hepatitis is generally diagnosed by serology and liver function tests

because liver biopsy is contraindicated. Chronic viral hepatitis is characterized by mononuclear portal infiltration. In chronic hepatitis B ground glass hepatocyte is the hallmark feature.[10] The presence of steatosis is significantly more common in patients with chronic hepatitis C than in patients with either chronic hepatitis B or autoimmune hepatitis.[11]

Hepatitis activity index (HAI) and fibrosing stage are two important findings during the evaluation of liver samples in patients with chronic hepatitis B, AIH is characterized by an irregular pattern of fibrosis and macronodular cirrhosis.[12]

HISTOLOGICAL GRADING OF NECROINFLAMMATORY ACTIVITY (HEPATITIS) OF PRIMARY BILIARY CIRRHOSIS	
HA 0 (no activity)	No interface hepatitis and no or minimal lobular hepatitis
HA 1 (mild activity)	Focal interface hepatitis and mild to moderate lobular hepatitis
HA 2 (moderate activity)	Between HA 1 and HA 3
HA 3 (marked activity)	Marked interface hepatitis, moderate to marked lobular hepatitis or bridging or zonal necrosis

Autoimmune hepatitis show extensive interface hepatitis, plasma cell predominance and hepatocyte rosettes in areas of marked activity.

Autoimmune cholangitis includes Primary biliary cirrhosis (PBC) and Primary sclerosing cholangitis (PSC). In PBC there are florid duct lesions and loss of small ducts. In PSC there is destruction of extrahepatic and large intrahepatic ducts, fibrotic obliteration of medium and small intrahepatic ducts.

Epidemiological studies suggest the prevalence of non alcoholic fatty liver disease (NAFLD) to be around 9-32% in general Indian population, with a

higher incidence amongst overweight/obese and diabetic/ prediabetic patients.[13] Both alcoholic hepatitis and NAFLD show steatosis, steatohepatitis and steatofibrosis even though features of steatohepatitis (e.g. hepatocyte ballooning, Mallory denk bodies, neutrophilic infilterates) are less prominent in NAFLD. The burden of alcohol-related disease is highest in the developed world, where it may account for as much as 9.2% of all disability-adjusted life years.[14] It has been concluded in a study by WHO that 90% of individuals who drink more than 60g/day of alcohol develop fatty liver.[15] Acute hepatitis present in 10-35% of alcoholic patients[16] and 5-15% progress to cirrhosis.[17-19]

In haemchromatosis there is deposition of golden yellow haemosiderin granules on cytoplasm of periportal hepatocytes that stain with Prussian blue. Wilson's disease is autoimmune disease having failure to incorporate copper into ceruloplasmin leading to hepatic injury.

End stage chronic liver disease is the most common indication of liver transplantion. Histopathology helps to examine the host liver to be removed at transplantation.[20] In transplanted liver, in acute rejection there is infiltration by mixed portal inflammatory infiltrates with bile duct injury and endothelitis. In chronic rejection there is ischemia of liver parenchyma due to obliteration of small and large arteries. Destruction of bile ducts leads to vanishing bile duct syndrome requiring retransplantation.

Hepatic Mass Lesions include benign cysts hemangioma, adenoma, liver abscess (amebic or pyogenic), focal nodular hyperplasia, fatty infiltration. The majority of these are classified according to the putative cells of origin (hepatocytes, bile duct epithelium and endothelium).[21]

6.3 AIMS AND OBJECTIVES OF THE STUDY

To study histopathological spectrum of non neoplastic liver diseases.

7.0 MATERIAL AND METHODS

All the trucut and wedge liver biopsies received from the various departments like gastroenterology, medicine and surgery in the department of pathology, Dayanand Medical College and hospital, Ludhiana . Tissue will be received in 10% formalin.Tissue processing will be done by embedding it in paraffin wax with the help of automated tissue processor.Then sectioning will be done using automated microtomes followed by staining with haematoxylin and eosin stain and special stains like masson trichome, reticulin, Periodic Acid Schiff (PAS), prussian blue, rhodanine, orcein etc. The slides will be subjected to histopathological examination under both low power (10X) and high power (40X).

7.1 SOURCE OF DATA

This is a 3 ½ years retrospective and 1 ½ years prospective study conducted for 5 years in the department of pathology, Dayanand Medical College and hospital, Ludhiana. All the biopsy specimen suspicious of non neoplastic liver disease received for histopathological examination will be included in the study.

7.2METHOD OF COLLECTION OF DATA

Inclusion criteria: All the trucut and wedge liver biopsies of non neoplastic liver diseases.

Exclusion criteria: Liver resections, explants specimen or biopsies with neoplastic etiologies.

Methodology: Liver biopsies will be received in container containing 10% formalin. Tissue will be processed and stained with heamtoxylin and eosin (H&E) stain. Special stains like masson trichome, reticulin, Periodic Acid

Schiff (PAS), prussian blue, rhodanine, orcein will be done wherever required. All the biopsy specimen received will be correlated with liver function tests which include bilirubin, SGOT, SGPT, GGT, albumin and prothrombin time.

Duration of study: Retrospective 3 ½ years and prospective 1 ½ years.

Study Design: This is prospective and retrospective study.

Outcome: All the findings of specimen will be documented according to the protocol.

Statistical Analysis: Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEF?

None.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Yes.

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PROFORMA

Serial no.-

Histopath No:

Name:

Age/Sex:

MRD No.

Date:

Address:

Phone No.

Chief Complaints:

Any history of Jaundice/Diabetes Mellitus/Viral Hepatitis/Alcohol/Obesity/Others

Clinical Examination-

Abdominal Mass/Hepatomegaly/Splenomegaly/Lymphadenopathy

Radiological Findings-

Bio-chemical Investigations-

LFTs

Viral Markers

Autoimmune markers

Ceruloplasmin

Iron stain

Gross: Wedge liver biopsy (Size)

Trucut liver biopsy (Length/ Number)

Microscopy:

Number of portal tracts:

Architecture: Lobular

Portal

Signature of Candidate

Signature of Supervisor

Fatty changes: Degree

Type

Cholestasis:

Bile: Infarct/Necrosis

Thrombi:

Stasis:

Lakes:

Bile Duct: Absent:

Distorted:

Reaction:

Mallory Hyaline/Mallory Denk Bodies:

Inflammatory Infilterates: Portal Tracts/ septa/ Sinusoids

Plasma Cell Rich:

Granuloma:

Interface Hepatitis:

Spotty Hepatic Necrosis:

Confluent Necrosis:

Fibrosis:

Nil

Grade 1

Grade 2

Grade 3

Grade 4

Stains: Iron

Masson Trichome:

Reticulin:

Others:

Impression:

Signature of Candidate **Signature of Supervisor**